ORIGINAL ARTICLE

Clinical manifestation of systemic lupus erythematosus in patients with antiribosomal P protein antibodies

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KEY WORDS

antibodies against ribosomal P protein, disease activity, systemic lupus erythematosus

ABSTRACT

INTRODUCTION Antibodies against ribosomal P protein (anti-P) are detected predominantly in patients with systemic lupus erythematosus (SLE). However, the data on their frequency and clinical relevance remain inconclusive.

OBJECTIVES The aim of the study was to assess the frequency as well as clinical and serological relevance of anti-P autoantibodies in Polish patients with SLE and to determine the significance of these antibodies in the diagnosis of SLE.

PATIENTS AND METHODS Anti-P antibody levels were measured in the sera of 100 SLE patients using an enzyme-linked immunosorbent assay and Western blotting. All patients underwent a routine clinical and laboratory evaluation. Disease activity was assessed using the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score.

RESULTS Anti-P antibodies were detected in 14 of 100 patients. When compared to anti-P-negative patients, this group was characterized by earlier onset of SLE, higher disease activity, more frequent occurrence of fever and facial erythema, decreased serum levels of complement, and elevated levels of alanine and aspartate aminotransferases. In 2 cases, anti-P antibodies were the only serological marker of SLE detected in these patients.

CONCLUSIONS SLE with the presence of anti-P antibodies is characterized by an early onset and high disease activity.

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Received: November 23, 2009.
Revision accepted: February 10, 2010.
Conflict of interests: none declared.
Pol Arch Med Wewn. 2010; 120 (3): 76-81
Translated by Anna Kalińska, MD

INTRODUCTION While several autoantibodies have been detected in patients with systemic lupus erythematosus (SLE), only antibodies against double stranded DNA (anti-dsDNA) and Sm antigen (anti-Sm) are considered to be SLE-specific. A similar role and clinical significance has been attributed to antibodies against ribosomal P protein (anti-P).[1-4]

Ribosomal P protein is a pentamer consisting of 3 different phosphoproteins forming monomer P0 and dimers P1 and P2. It plays an important role in all stages of protein synthesis. Anti-P antibodies recognize at least 1 epitope common for all 3 phosphoproteins and are reactive with linear structure of antigenic determinant within their homologous C-terminal 22 amino acid sequence.[5] The presence of anti-P antibodies might be associated with multiple organ involvement in the course of SLE, including the central nervous system (CSN),[6-9] kidneys,[10-12] or liver,[13-15] but the results from various studies are still conflicting.

The aim of this study was to assess the prevalence of anti-P antibodies among Polish patients with SLE, to characterize the clinical manifestation of this disease in a group of anti-P positive patients, and to define the role of anti-P in the diagnosis of SLE.

PATIENTS AND METHODS The study included 100 women with SLE, consecutively admitted to

TABLE 1 Characteristics of SLE patients

Parameter	Anti-P/–/ (n = 86)	Anti-P/+/ (n =14)	*P*
age at disease onset (years)	35.2 ±15.2[a] median: 33 (min. 10 – max. 70)	27.9 ±16.4[a] median: 23.5 (min. 11 – max. 70)	<0.05
<5 years	4 (5%)	4 (29%)	<0.05
16–44 years	58 (68%)	8 (57%)	
>45 years	23 (27%)	2 (14%)	
age at time of study (years)	40.8 ±14.9[a] median: 41 (min. 18 – max. 76)	33.6 ±16.0[a] median: 28 (min. 19 – max. 70)	<0.05
disease duration (months)	68.7 ±8.1[b] median: 48 (min. 2 – max. 312)	82.1 ±25.4[b] median: 66 (min. 4 – max. 360)	NS
<3 years	41 (48%)	6 (43%)	NS
3–9 years	27 (32%)	3 (21%)	
>9 years	17 (20%)	5 (36%)	

a standard deviation
b standard error of mean

Abbreviations: NS – nonsignificant, SLE – systemic lupus erythematosus

the Department of Connective Tissue Diseases at the Institute of Rheumatology in Warsaw, Poland. SLE diagnosis was based on the classification criteria of the American College of Rheumatology, which were established in 1982 and subsequently modified in 1997.[16,17]

All patients underwent a medical examination and routine laboratory evaluation (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP], peripheral blood cell counts, protein electrophoresis, biochemical tests). We also performed additional tests including electrocardiography, chest X-ray, abdominal ultrasound, etc.

Several immunological parameters were assessed in each patient:

1 antinuclear antibodies (Colorzyme, Immuno-Vision, United States) tested on fixed Hep-2 cells

2 anti-dsDNA antibodies assayed by an enzyme-linked immunosorbent assay (ELISA; Pharmacia Diagnostics, Germany)

3 antibodies against soluble nuclear antigens, including anti-Sm, anti-RNP, anti-Ro (SSA, Sjögren's syndrome A antibody), anti-La (SSB, Sjögren's syndrome B antibody), as well as antihistone and antimitochondrial antibodies tested with INNO-LIA® ANA Update (INNOGENETICS, Belgium)

4 anticardiolipin antibodies assayed by ELISA according to the modified method by Gharavi[18]

5 immunoglobulin (Ig) M rheumatoid factor and C3 complement component determined using nephelometry

6 anti-P antibodies tested by an ELISA (EUROIMMUN, Germany) as a screening test, and by Western blotting as a confirmation test.

The Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score was used to assess disease activity.[19]

Statistical analysis Statistical analysis was performed using the statistical package SPSS/PC+. In the first stage of analysis, compliance of continuous random variables with Gaussian curve was analyzed using the Kolmogorov-Smirnov test. Statistical significance of differences between averages for parameters of normal distribution was assessed using the Student's t-test, and for parameters deviating from normal distribution the nonparametric Mann-Whitney test and the median test were used.

In the second stage, we used the χ^2 Pearson test or Fisher's exact test to assess frequency differences of specified levels of qualitative variables, presented in nominal scales. The results of these variables are presented as proportions.

We assumed the level of statistical significance at $P < 0.05$. All analyzed tests were bilateral.

RESULTS On the basis of the anti-P antibody measurement, 100 patients included in our study were classified into 2 groups, namely 86 patients with the negative result of anti-P tests (anti-P/–/) and 14 patients with the positive result (anti-P/+/). TABLE 1 presents the characteristics of both patient groups.

Higher incidence of fever (51% vs. 86%, $P < 0.05$) and malar rash (49% vs. 86%, $P < 0.05$) was observed in the anti-P/+/ group. Involvement of the nervous system was observed in 22 anti-P/–/ patients (psychoorganic syndrome in 2, depression in 8, convulsions in 4, stroke in 5, cranial nerves neuropathy in 2, polineuropathy in 1) and in 2 anti-P/+/ patients (depression in 1, stroke in 1) (*P* = nonsignificant [NS]).

The 2 groups did not differ significantly with respect to either peripheral blood cell counts or the laboratory indicators of inflammation (ESR, CRP, α_2-, γ-globulin) (*P* = NS).

Interestingly, anti-P/+/ patients had significantly higher levels of alanine and aspartate aminotransferases (9.5% vs. 36%, $P < 0.05$). However, this increase was moderate and did not exceed twice the upper limit of the normal value. In isolated cases, an increase in aminotransferases was accompanied by elevated alkaline phosphatase and γ-glutamyltransferase. Moreover, routine abdominal ultrasound revealed enlarged liver and enhanced liver echogenicity in 3 anti-P/–/ and 2 anti-P/+/ patients.

Alcohol abuse, infection, and pharmacological factors were excluded as possible causes of these symptoms. Antimitochondrial antibodies were negative in all studied patients.

Increased aminotransferases were detected in both (2 out of 2) anti-P/+/ patients displaying signs or symptoms of the CNS involvement. In comparison, such increase was observed in only 2 out of 22 anti-P/–/ patients with neuropsychiatric symptoms ($P < 0.05$).

Various antinuclear antibodies were positive in the majority of patients. Anti-P antibodies were accompanied most frequently by anti-dsDNA, anti-RNP, anti-Sm, and antihistone antibodies,

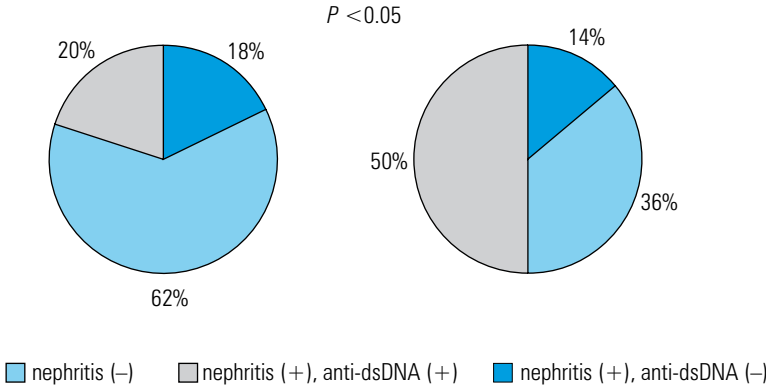

FIGURE 1 Concomitant lupus nephritis and anti-dsDNA antibodies in anti-P/–/ and anti-P/+/ patients

but none of these correlations achieved statistical significance (P = NS).

A significant decrease of C3 complement component was observed in anti-P/+/ patients (71% vs. 29%, P <0.05), while kidney involvement was associated more often with anti-dsDNA antibodies (50% vs. 22%, P <0.05) (FIGURE 1).

In order to establish the diagnostic value of anti-P antibodies in SLE, we analyzed the frequency of marker antibodies (according to point 10 of SLE diagnostic criteria). Among 14 anti-P/+/ patients only 1 displayed all 4 types of autoantibodies, 4 patients had anti-dsDNA and anti-Sm antibodies, 6 patients had only anti-dsDNA. There were a few cases of single antibody production, namely only anti-Sm or only antiphospholipid antibodies. Two anti-P/+/ patients did not display any of the autoantibodies listed in point 10 of the diagnostic criteria.

The majority of anti-P/+/ patients exhibited high disease activity (>20 points SLEDAI; 79% vs. 44%, P <0.05) (FIGURE 2). Analysis of data by median test indicated that for 50% of anti-P/+/ patients disease activity was above 23 points SLEDAI, while 50% of anti-P/–/ patients had SLEDAI score above 18 (P <0.05) (TABLE 2).

Comparative analysis of SLE activity in both groups at the onset of the disease and during the course of this study revealed that while in the anti-P/–/ patients, the initially high disease activity eventually decreased significantly, in the anti-P/+/ group it remained high.

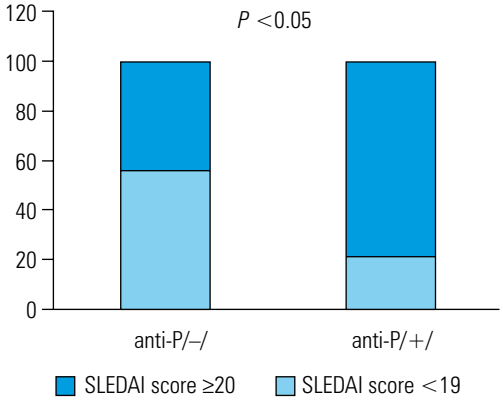

FIGURE 2 Activity of systemic lupus erythematosus as measured by the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI)

TABLE 2 Disease activity

Parameter	Anti-P/–/	Anti-P/+/	P
SLEDAI score min. – max.	18.3 ± 9.7 median: 18 (4–49)	23.1 ± 6.7 median: 23.5 (12–37)	<0.05
<19	48 (56%)	3 (21%)	<0.05
≥20	37 (44%)	11 (79%)	

Abbreviations: see FIGURE 2

DISCUSSION Although discovered over 30 years ago, anti-P antibodies are still somewhat underestimated in the diagnosis of SLE, and there is no consensus regarding their correlation with clinical picture and their role in the pathogenesis of this disease.

Genetic and environmental factors are most likely responsible for the discrepancies in reported frequencies of anti-P in SLE patients. For example, 30% to 42% of SLE patients within Chinese and Japanese populations are anti-P positive.[20-22]

Frequency of anti-P detected in this study reached 14%, which is in agreement with other studies conducted in the white population.[22,23]

Strikingly, these antibodies are quite common in pediatric patients and young adults with SLE, reaching 20% to 42%.[23-25] In line with these studies, almost 30% of our anti-P/+/ patients developed SLE before the age of 15, and half before the age of 23. In contrast, 50% of patients in the anti-P/–/ group developed the disease before the age of 33, which represents a statistically significant difference (FIGURE 3).

Fever, skin lesions, and arthritis appeared to be the prevailing symptoms in our patients and were detected significantly more often in the anti-P/+/ group (P <0.05). Other authors reported even more frequent skin involvement in anti-P/+/ patients, particularly malar rash, discoid lupus erythematosus, sensitivity to ultraviolet light, and cutaneous manifestations of vasculitis.[9,23,26]

Concomitant occurrence of lupus nephritis and anti-dsDNA antibodies was twice as frequent in patients with anti-P/+/ as in anti-P/–/ group, similarly to the results reported previously by others (FIGURE 1).[10-12]

In 2 of the studied patients, serological abnormalities were limited to anti-P antibodies. As reported by other authors, 35% of anti-P/+/ SLE patients do not have anti-dsDNA antibodies.[27-29] It is postulated that anti-P can be a valuable serological marker of SLE and play an important role in a diagnostic process, being especially helpful in cases of SLE fulfilling any 4 diagnostic criteria with the exception of point 10, i.e., lack of marker antibodies.

In the present study, similarly to the previously published data, there was no relationship between anti-P and the CNS involvement.[30]

The results of several studies suggest higher frequency of lupoid hepatitis in patients with anti-P antibodies.[11,13] The term "lupoid hepatitis" was first used by I.R. Mackay in 1959 to describe chronic progressive hepatitis accompanied by serological disturbances typical for SLE.[31] Currently,

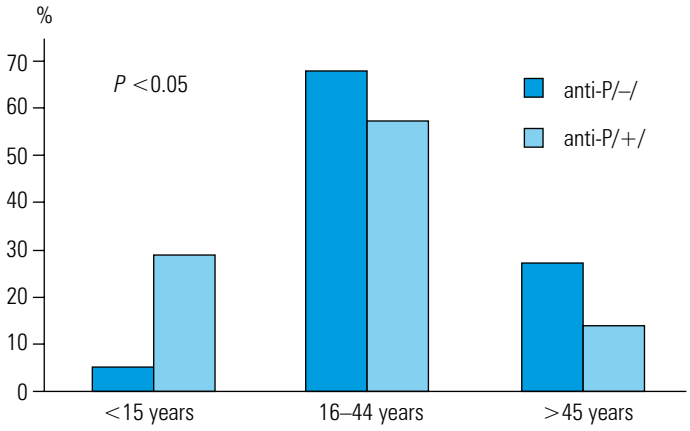

FIGURE 3 Onset of the disease in patients with systemic lupus erythematosus within different age groups

this disease is classified as chronic active autoimmune hepatitis with antinuclear antibodies. Typically, autoimmune non-SLE-related chronic active hepatitis patients do not have anti-P antibodies,[11] suggesting a potential use of anti-P in diagnostic differentiation of these 2 types of hepatitis.

Indeed, our experience confirms such a possibility, since the increased levels of liver enzymes were detected in 36% of anti-P/+/ patients and only in 9.5% of anti-P/–/ patients. Moreover, in agreement with previous reports, we observed a link between neurological symptoms, an increase in aminotransferases and the presence of anti-P.[11,32]

It was possible to gain better understanding of this correlation in the light of the studies by Koren et al.[33] and Koscec et al.[15] Ribosomal P protein has been reported to be expressed on the surface of cultured human *hepatoma* and *neuroblastoma* cells; furthermore, it has been confirmed that anti-P antibodies are able to bind to *hepatoma* cells, penetrate into live hepatocytes, and cause cellular dysfunction in vitro.

Anti-P antibodies are detected more frequently in SLE patients with concomitant anti-dsDNA and anti-Sm antibodies. It was demonstrated that both anti-P and anti-dsDNA antibodies are able to bind not only ribosomal P protein but also deoxyribonucleic acid. Anti-dsDNA antibodies have a cytotoxic potential and are sensitive to at least partial blocking with C-terminal peptide derived from protein P, or with recombinant protein P1. Since the expression of protein P is ubiquitous (fibroblasts, endothelial cells, lymphoid cells, liver and kidney cells) such interaction with anti-dsDNA antibodies may indeed play a significant role in the pathogenesis of SLE.[34,35] On the other hand, anti-P proved to be cross-reactive with other antibodies, such as anti-Sm and antiphospholipid.[36,37]

Activity of SLE measured by the SLEDAI score was significantly higher among anti-P/+/ patients.[9,40] Comparative analysis of SLE activity in both groups at the onset of the disease and during this study revealed that it remained high in the anti-P/+/ group of patients despite the treatment. It may imply more severe course of the disease and poorer response to therapy, especially when compared with the anti-P/–/ group, in which the initially high disease activity eventually decreased.

Anti-P antibodies constitute an important indicator of organ damage and disease activity in SLE. Detected mainly in SLE patients, these antibodies certainly deserve to be considered as markers of this disease as well as a valuable tool in diagnostic process. Results from experimental studies further corroborate their involvement in the pathogenesis of SLE.

Acknowledgments The study was supported by the Ministry of Science and Higher Education grant no. 2 P05B

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ARTYKUŁ ORYGINALNY

Obraz kliniczny tocznia rumieniowatego układowego u chorych z przeciwciałami przeciw rybosomalnemu białku P

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SŁOWA KLUCZOWE

aktywność choroby, przeciwciała przeciw rybosomalnemu białku P, toczeń rumieniowaty układowy

STRESZCZENIE

WPROWADZENIE Przeciwciała przeciw rybosomalnemu białku P (anty-P) stwierdza się głównie u chorych na toczeń rumieniowaty układowy (*systemic lupus erythematosus* – SLE), ale dane na temat częstości ich występowania i korelacji klinicznych są niejednoznaczne.

CELE Celem badania była ocena częstości występowania, powiązań klinicznych i serologicznych przeciwciał anty-P u polskich chorych ze SLE oraz określenie ich znaczenia w ustaleniu rozpoznania SLE.

PACJENCI I METODY Przeciwciała anty-P oznaczono w surowicy 100 chorych na SLE za pomocą testu immunoenzymatycznego i Western blot. U wszystkich chorych przeprowadzono badanie lekarskie oraz rutynowe badania laboratoryjne. Aktywność choroby oceniono za pomocą skali SLEDAI (*Systemic Lupus Erythematosus Disease Activity Index*).

WYNIKI Przeciwciala anty-P wykryto u 14 spośród 100 chorych. W porównaniu z chorymi bez przeciwciał anty-P, grupa ta charakteryzowała się początkiem SLE w młodszym wieku, większą aktywnością choroby, częstszym występowaniem gorączki, rumienia na twarzy, hipokomplementemią oraz wzrostem stężenia transaminazy alaninowej i asparaginianowej. U 2 chorych przeciwciała anty-P były jedynym markerem serologicznym SLE.

WNIOSKI SLE z obecnymi przeciwciałami anty-P cechuje się początkiem choroby w młodym wieku oraz wysoką aktywnością choroby.

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Praca wpłynęła: 23.11.2009.
Przyjęta do druku: 10.02.2010.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2010; 120 (3): 76-81