

Raynauds phenomenon, peripheral neuropathy, renal involvement and thrombocytopenia, was found to be important overall for discriminating SLE patients with or without SS. SLEwSS patients constitute a subgroup of patients with SLE characterized by milder lupus: older age at death, similar rates of mortality and SLICC-ACR damage index, less renal and immunological manifestations.

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PERSISTENCE OF ANTI-SMITH ANTIBODY IS ASSOCIATED WITH DISEASE ACTIVITY IN PATIENTS WITH NEW-ONSET SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Anti-Smith (Sm) antibody is highly specific antibody for systemic lupus erythematosus (SLE). We evaluated the association between anti-Sm antibody and disease activity in patients with new-onset SLE.

Methods Patients who were repeatedly tested for anti-Sm antibody at SLE diagnosis and within 12 months were included in this study. The clinical and laboratory profiles, and systemic lupus erythematosus disease activity index (SLEDAI) were collected at the time of anti-Sm antibody test. SLEDAI and laboratory variables associated with disease activity were compared between patients with and without anti-Sm antibody.

Results Of 92 patients who were tested for anti-Sm antibody at SLE diagnosis, 67 patients were followed up for presence of anti-Sm antibody at 6 months, and 67 patients were followed up at 12 months. Although the baseline SLEDAI was comparable in SLE patients with or without anti-Sm antibody, immunologic and hematologic disorder was more common in anti-Sm positive patients. Patients who showed positive result of anti-Sm antibody at 6 and 12 months had higher SLEDAI compared to patients with negative result ($p=0.004$ and 0.002 at 6 and 12 months, respectively). The changes in anti-Sm antibody for 12 months was significantly correlated with the changes of SLEDAI ($p=0.029$).

Conclusions Persistence of anti-Sm antibody for 12 months was associated with higher disease activity at the corresponding time. Follow-up of anti-Sm antibody can be useful to evaluate the remained disease activity in patients with new-onset SLE.

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CLINICAL CHARACTERISTICS OF PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS SHOWING THE FALSE POSITIVE RESULT OF SYPHILIS TEST

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Background False positive result of syphilis test is a characteristic finding in patients with systemic lupus erythematosus (SLE), especially combined with antiphospholipid syndrome (APS). We evaluated the clinical characteristics in SLE patients who showed the false positive result of syphilis test.

Methods Patients who were tested for syphilis screening test at SLE diagnosis in Severance Hospital between January 2006 and December 2016 were included in this study. The baseline characteristics and clinical outcomes were compared between

patients with false positive result of syphilis test and negative result of syphilis.

Results Of 145 patients included in this study, 20 (13.8%) patients showed the false positive result of syphilis test. At SLE diagnosis, patients with negative syphilis result had higher SLE disease activity index (5.0 vs 8.0, $p<0.001$), and were more commonly complicated with nephritis (15.0% vs 41.6%, $p=0.026$). Low disease activity, high protein level, and presence of APS antibodies were independently associated with the false positive result of syphilis test ($p=0.030$, 0.014 and 0.002 , respectively). Although the thrombotic risk was significantly higher in patients with false positive syphilis result ($p=0.041$), the overall mortality showed no difference between patients with false positive result and negative result of syphilis test.

Conclusions Clinical characteristics of SLE patients with false positive result of syphilis test showed lower disease activity at SLE diagnosis, but comparable overall survival and higher thrombotic risk.

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ANTIRIBOSOMAL P AUTOANTIBODIES TARGET THE NEURONAL-SURFACE-P-ANTIGEN (NSPA) IN KIDNEY AND LIVER CELLS

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Background Anti-ribosomal P (anti-P) antibodies have long been associated with lupus psychosis and recently with cognitive deficit in patients with systemic lupus erythematosus (SLE). We previously described a neuronal-surface-P-antigen (NSPA) that mediates anti-P pathogenic effects leading to memory deficit in mice. Clinical controversial associations of anti-P are lupus hepatitis and lupus nephritis (LN), in which the ribosomal P0 protein has been postulated as a cell surface anti-P target. As there is no mechanism explaining for the presence of P0 at the cell surface, we assess whether NSPA might be the anti-P cell surface target in liver and kidney cells.

Methods NSPA expression: i) RT-PCR and immunoblot in liver HepG2 and kidney HK-2 cell lines and liver and kidney tissues from C57wt and transgenic C57NSPA/KO (LacZ gene instead NSPA gene) mice; ii) β -galactosidase histochemistry as a marker of NSPA expression in liver and kidney slices from a ZZEF-1/lac Z knock in mice; iii) Cell surface biotinylation and surface immuno-capture in combination with metabolic labeling. Functional assays: iv) Internalization assays with I125-anti-P; v) Indirect immunofluorescence of activated caspase-3 and Hoechst staining of apoptotic nuclei on HK-2 treated with rabbit anti-P for 24 hour.

Results NSPA is expressed in hepatocytes, in proximal epithelium tubule renal cells and in some collecting tubules. HK-2 and HepG2 express NSPA, but not P0 at the cell surface. Anti-P bind NSPA and become internalized in a time and concentration dependent manner. Anti-P induced HK-2 apoptosis *in vitro* assessed by caspase-3 activation.

Conclusions NSPA expression in the cell surface of kidney and liver cells and not the P0 provides a potential target for anti-P pathogenic effects, which might contribute to lupus hepatitis and nephritis.

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TREATMENT OUTCOME IN LUPUS NEPHRITIS PATIENTS TREATED WITH MYCOPHENOLATE MOFETIL: FROM A REAL-WORLD CLINICAL PRACTICE

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Background Systemic lupus erythematosus (SLE) is autoimmune disorder often characterized by the development of glomerulonephritis. The use of mycophenolate mofetil (MMF) is highlighted as induction and maintenance therapy in lupus nephritis. We evaluated the treatment outcome of MMF in lupus nephritis patients from a real clinical practice.

Methods Patients with biopsy proven lupus nephritis (class III, IV, and V) between November 2005 and August 2017 in Severance Hospital were extracted, and those patients who were treated with MMF at least 3 months were included in this study. The remission rate of lupus nephritis and risk factors for failure of remission were evaluated using Kaplan-Meier analysis and Cox proportional hazards model.

Results Of 116 patients included in this study, 89 (76.7%) patients achieved remission of lupus nephritis after treatment with MMF. The median time to remission was 4.2 months (interquartile range 0.9 9.1). Normal complement level, negative result of anti-dsDNA antibody, and nephrotic range proteinuria were risk factors for remission failure in univariate analysis ($p=0.017$, 0.001 , and 0.007 , respectively). Nephrotic range proteinuria and negative result of anti-dsDNA antibody are independently associated with remission failure in multivariate analysis (OR 3.19, $p=0.004$ and OR 1.62, $p=0.028$, respectively).

Conclusions Patients with lupus nephritis showed a favourable clinical outcome after MMF treatment. However, additional therapy would be required in patients with nephrotic-range proteinuria and without anti-dsDNA antibody.

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ANTI-RIBOSOMAL P AUTOANTIBODIES ARE NOT A MARKER FOR LUPUS NEPHRITIS

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Background Studies looking for clinical association of anti-ribosomal P (anti-P) autoantibodies and lupus nephritis (LN) describe contradictory results. It is clear that anti-dsDNA antibodies contribute to LN pathogenesis and their titers fluctuate together with those of anti-P, suggesting a linked generation

Abstract 126 Table 1 Lupus nephritis histology class and anti-dsDNA and anti-P presence

| | Autoantibodies | | | |
|--|----------------------------------|-------------------------------|------------------------------|-----|
| | Anti-dsDNA present and no anti-P | Anti-dsDNA and anti-P present | Anti-dsDNA and anti-P absent | |
| Lupus Nephritis Class | n=26 | n=20 | n=4 | n=2 |
| ISN/RPS | | | | |
| Proliferative Class III or IV | 17 | 14 | 3 | 0 |
| Mixed Proliferative Class III or IV and Membranous Class V | 7 | 5 | 1 | 1 |
| Membranous Class V | 2 | 1 | 0 | 1 |
| "pure" | | | | |

or an anti-dsDNA cross-reaction with the P antigen. We reexamined the anti-P involvement in LN in relation with the possibility of anti-dsDNA and anti-P cross-reactivity

Methods Anti-P and anti-dsDNA were analyzed by ELISA. SLE sera ($n=24$) from patients with and with no LN were divided into 4 groups: A (anti-dsDNA positive, anti-P negative), B (both positive), C (anti-dsDNA negative, anti-P positive) and D (both negative). Anti-dsDNA cross-reaction was assessed against recombinant wild type and P-epitope-lacking P0 proteins using purified IgGs from SLE patients. Anti-P cross-reaction with dsDNA was analyzed testing affinity purified anti-P antibodies with an anti-dsDNA ELISA. LN biopsies ($n=26$) were classified according to ISN/RPS and relations to the anti-dsDNA and anti-P simultaneous presence were examined

Results Neither anti-dsDNA nor anti-P antibodies showed evidence of cross-reaction in any of the applied tests. LN biopsy proven patients: age (media \pm SD) 34 ± 11 years; 88% female. No LN histologic class with anti-P relationship was observed: 9 patients had Class V (pure $n=2$) or mixed with Class III/IV ($n=7$); 1 patient was anti-P positive (p value >0.05). Two patients with LN had neither anti-dsDNA nor anti-P antibodies. The NIH Activity Index (8.9 ± 4.9) and NIH chronicity index scores (1 ± 1.1) and tubule-interstitial lesions were similar between anti-P positive or negative LN patients

Conclusions Cross-reactivity between anti-dsDNA and anti-P antibodies cannot explain the contradictory results of anti-P association with LN. Based on the present and our previous studies failing to find anti-P association with LN, it seems unlikely that anti-P contribute to the renal damage. Other factors beyond anti-dsDNA and anti-P might participate in LN

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HEPATIC INVOLVEMENT AS A PRESENTATION IN PEDIATRIC LUPUS: A RETROSPECTIVE STUDY OF 3 CASES

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Background Though abnormal liver tests can be seen during the course of disease in lupus, liver involvement as a