

Methods Pristane was injected in female BALB/c mice to induce the disease. After five months, mice in various groups were treated with prednisone and fed with gluten containing and standard diet for four weeks and applied procedure to detect minor changes in paw swelling, ANA autoantibodies, CCL11, C3c, glucose level and renal damage.

Results We detected increased symptoms of arthritis and gastrointestinal tract involvement in gluten containing diet group compared with standard diet disease control group. ANA autoantibodies, C3c and renal damage between gluten and standard diet group was non-significant. The remission of SLE manifestations was observed in prednisone treated group except renal damage.

Conclusions From the study it was concluded that gluten intake could worsen the clinical manifestations in SLE patients, therefore, administration of gluten free diet might be a better strategy for SLE patients. However, further confirmatory studies are required in this regard.

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SLE RISK HAPLOTYPES ARE ASSOCIATED WITH DEVELOPMENT OF SEROLOGIC AUTOIMMUNITY IN HEALTHY INDIVIDUALS

¹P Raj, ²D Karp*, ¹I Dozmorov, ¹Q Li, ¹E Wakeland. ¹UT Southwestern Medical Centre, Immunology, Dallas, USA; ²UT Southwestern Medical Centre, Rheumatic Diseases Division, Dallas, USA

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Background and aims ANA are one of the earliest features of lupus, preceding the onset of clinical symptoms. The genetic risk factors that underlie the development of serological autoimmunity are unknown. A genome-wide association study was undertaken to understand the genetics of ANA development.

Methods Serum and DNA were collected from 2635 healthy individuals with no personal history of autoimmunity. Sera from 724 individuals (ANA-, ANA+, and SLE) were assayed by protein microarray quantifying IgM and IgG responses to 96 human autoantigens. A nested cohort of subjects consisting of all the ANA+ Caucasian individuals and matched ANA-controls were genotyped.

Results In healthy individuals, 16.2% had moderate and 8.0% had high levels of IgG antinuclear antibodies. ANA+ healthy individuals had a high prevalence of antibodies to non-nuclear and cytoplasmic antigens, while subjects with SLE predictably produced antibodies to a variety of nuclear antigens. A quantitative genetic association test with ANA identified genomic loci associated with high ANA phenotype. *HLA* was second strongest signal ($p=6.2 \times 10^{-6}$). The frequencies of SLE risk haplotypes at several loci were significantly increased in the ANA high positive group compared to ANA negative subjects. However, SLE risk haplotypes at other loci were only high in the SLE group, suggesting their main role in a transition to clinical disease.

Conclusions The genetic risk for the development of ANA includes many of the previously documented SLE risk haplotypes. However, other genetic associations are specific for SLE, suggesting distinct risk factors for ANA and for lupus.

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CLINICAL USEFULNESS OF QUANTITATIVE MEASUREMENT OF ANTI-M-TYPE PHOSPHOLIPASE A2 RECEPTOR ANTIBODIES IN PATIENTS WITH MEMBRANOUS NEPHROPATHY AND COMPARISONS OF THREE QUANTITATIVE METHODS

R Moriyama*, Y Katsumata, Y Okamoto, Y Kawaguchi, H Yamanaka. Tokyo Women's Medical University, Institute of Rheumatology, Tokyo, Japan

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Background and aims Autoantibodies to M-type phospholipase A2 receptor (PLA2R) are specific markers of idiopathic membranous nephropathy (MN). It has also been suggested that anti-PLA2R antibody is associated with disease activity and prognosis but more solid evidence is needed. We aimed to establish quantitative measurement of anti-PLA2R antibodies and further investigate its clinical usefulness.

Methods Using stable cell line expressing PLA2R, we developed a quantitative cell-based enzyme-linked immunosorbent assay (ELISA) and Western blot (WB) for anti-PLA2R antibodies. The usefulness of these tests and the commercial solid phase ELISA were retrospectively studied in sera from 23 patients with biopsy-proven primary MN, and 16 patients with lupus MN. Repeated sera were also available in 9 patients with primary MN.

Results Anti-PLA2R antibodies were detected in 12, 6, and 12 out of 23 patients with primary MN by the WB, the cell-based ELISA, and the commercial solid phase ELISA, respectively. Conversely, all of the samples from the lupus MN patients were negative. The levels of proteinuria were moderately correlated with titers of anti-PLA2R antibodies by the 3 methods ($r=0.39$ to 0.47). Anti-PLA2R antibodies were significantly associated with physicians' decision on immunosuppressive therapy without prior knowledge of anti-PLA2R antibody positivity ($p<0.01$). In all of the 6 patients who were treated with immunosuppressive therapy, titers of anti-PLA2R antibodies significantly declined by commercial solid-phase ELISA ($p=0.03$).

Conclusions This study showed that anti-PLA2R antibody is clinically useful as diagnostic and surrogate biomarkers in primary MN. In addition, the 3 methods are all reliable measurement methods for anti-PLA2R antibodies but demonstrated different performance.

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NAILFOLD VASCULAR FINDINGS OF ANTI-MELANOMA DIFFERENTIATION-ASSOCIATED GENE 5 ANTIBODY-POSITIVE PATIENTS WITH DERMATOMYOSITIS

¹K Komura*. ¹Tsuruga Hospital, Dept of Dermatology, Tsuruga, Japan

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Background and aims Rapidly progressive interstitial lung disease is complicated in majority of patients with dermatomyositis who are positive for anti-melanoma differentiation-associated gene 5 antibody (MDA5). Clinically amyopathic dermatomyositis with Heliotrope rash and Gottron's sign can offer an implication for MDA5-positivity and the critical treatment before uncovering the result of blood testing, since these patients can survive only if they received immediate and intensive therapy. We observed nailfold capillary formation of two acute patients with anti-MDA5 antibody using dermatoscopy in the current study in order to test the capability to predict the MDA5 positivity.