

SLEDAI at active and remission phases were 4.25 *vs* 0.45 and 8.32 *vs* 1.25, respectively. Fever, rash, and arthritis were the most common features and kidney was the most common involved organ at active phase. The mean serum complement factor H and I levels at active phase were significantly lower than that at remission phase. The mean serum CD46 level at active phase was higher significantly compared with that at remission phase. The serum C5a and C5b-9 at active and remission phases were no significant difference. Five patients had sequelae including 1 intracranial haemorrhage and 4 chronic kidney disease.

Conclusions Serum complement factor H, I and CD46, but not C5a and C5b-9 were associated with disease activity of SLE.

280 THE ROLE OF PI3K, MTOR IN THE EXPRESSION OF INTERFERON -ALPHA INDUCED PROTEIN IFIT4 IN LUPUS

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10.1136/lupus-2017-000215.280

Background and aims The role of Phosphoinositide 3-kinase (PI3K), mammalian target of rapamycin (mTOR) and dexamethasone in IFN- α -induced-human interferon-induced protein with tetratricopeptide repeats 4 (IFIT4) expression was investigated.

Methods HT1080 cells were pre-treated with specific inhibitors of PI3K/mTOR, PKC or JNK transduction factors, then further incubated with IFN- α for different times. The mRNA and protein expression of IFIT4 or other indicated signal transduction factors were detected by qRT-PCR or western-blot.

Results LY294002, a dual mTOR and PI3K inhibitor, but not wortmannin, blocked IFIT4 promoter activation, mRNA and protein, as well as phosphorylation of STAT1, JNK, PKC δ induced by IFN- α . Interestingly, rapamycin, mTOR inhibitor, had the same effects as LY294002, counteracting the IFN- α -dependent upregulation of IFIT4 and phosphorylation of STAT1, JNK, PKC δ . Rottlerin or Sp600125, specific inhibitor of PKC δ , JNK, inhibited IFN-induced IFIT4 expression, but not the phosphorylation of AKT and mTOR. Interestingly, *in vivo*, bolus intravenous injection of methylprednisolone rapidly decreased the IFIT4 expression. *In vitro*, dexamethasone could prohibit IFN- α -induced IFIT4 transcription and the phosphorylation of STAT1, JNK, PKC- δ .

Conclusions IFN- α activate the PI3K and mTOR pathways, which converge to regulate PKC δ , JNK, STAT1-dependent transcription of IFIT4 in a mTOR dependent and AKT independent manner. The induction of IFIT4 transcription by IFN- α depends upon sequential activation of mTOR, PKC δ , JNK and STAT1. Steroid might play the role in treatment for systemic lupus erythematosus (SLE) partially by the reason of decreasing IFN alpha induced protein IFIT4 expression via sequential inhibition of the phosphorylation of PI3K, mTOR, PKC δ , JNK, STAT1.

281 THE CLINICAL ANALYSIS OF THE CHARACTERISTICS OF LUPUS MESENTERIC VASCULITIS (LMV) COMPARED WITH LUPUS NEPHRITIS

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10.1136/lupus-2017-000215.281

Background and aims Evaluate the clinical characteristics of lupus mesenteric vasculitis (LMV) in a large cohort.

Methods The in-patients with Systemic Lupus erythematosus (SLE) admitted to the West China Hospital from 2009 to 2015 were analysed. Diagnosis of LMV was made according to the clinical symptoms, abdominal image and the reaction to steroids. The patients with Lupus Nephritis (LN) were set as the control. Qualitative differences were analysed using the chi-square test.

Results Among 3143 patients with SLE, 103 patients were diagnosed as LMV with the incidence of LMV being 32.8%. Among those patients with LMV, 96.1% was female and the average age was 35.8 years old, 37 (35.9%) patients were diagnosed as SLE with LMV as the one of onset symptoms, 54 (52.4%) patients were misdiagnosed at the first time. The incidence of abdominal pain was 87.4%, while that of pelvic effusion 68.04%, vomiting 70.9%, bloating 68.6% and diarrhoea 67.0%. Their average score of the SLE disease activity index (SLEDAI) (including LMV as vasculitis) was 21.8 ± 7.7 . During the following-up for 2–96 months, partial remission of the incidence of abdominal pain within a week was 87.9%, and 98.8% of the LMV patients got complete remission in a month. Eight cases experienced disease relapse (8.0%). Rash ($X^2=6.7255$, $p=0.0095$), high creatinine ($X^2=6.746$, $p<0.0001$), hypercholesterolemia ($X^2=21.2986$, $p<0.0001$), leukopenia ($X^2=7.4153$, $p=0.0245$) were presented more often in the patients with LMV than in LN.

Conclusions There were differences between LMV and LN in clinical symptoms and laboratory findings. A much lower mortality rate was found in LMV.

282 EFFECT OF GLUTEN CONTAINING DIET ON PRISTANE INDUCED LUPUS PRONE MICE

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10.1136/lupus-2017-000215.282

Background and aims SLE is a chronic autoimmune disease with characteristic organ involvement and autoantibodies production. The pathogenicity and aetiology of the disease yet to be elucidated. It is presently accepted that environmental factors trigger the disease in genetically sensitive individuals. Gluten, a protein fraction commonly found in wheat grains, associated with food related disorders and a number of autoimmune diseases. We hypothesised that gluten containing diet would further exacerbate an already undergoing arbitrary immune reaction in SLE patients.

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.

283

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

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10.1136/lupus-2017-000215.283

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.

284

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

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10.1136/lupus-2017-000215.284

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.

285

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

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10.1136/lupus-2017-000215.285

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.