

disease activity and active disease, there was no difference in BAFF/APRIL levels between groups. Serum BAFF levels were higher in patients with renal disease activity (median 0.94 ng/ml vs 0.61 ng/ml, $p=0.01$), and there was a positive correlation between APRIL levels and proteinuria ($r=0.42$, $p=0.02$). There was no association between BAFF/APRIL levels and anti-dsDNA positivity but a weak inverse correlation was observed between BAFF and C3 levels ($r=0.25$, $P=0.02$). No correlation was found between BAFF/APRIL levels and renal SLEDAI scores, histopathologic activity and chronicity index scores. In the active disease group after follow-up, there was no significant changes in BAFF (from 1,63 ng/ml to 1,2 ng/ml) and APRIL levels (from 2,11 ng/ml to 2,31 ng/ml).

Conclusions BAFF/APRIL levels were found to be significantly higher in patients with SLE compared to controls, but no association with disease activity was found. BAFF levels are correlated with decreased C3 levels. These results suggest that both cytokines are involved in the pathogenesis of SLE, and that serum BAFF and APRIL levels can be valuable biomarkers in SLE especially in patients with renal activity. Long-term studies on the effect of treatment are needed.

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P27 MYXOVIRUS RESISTANCE PROTEIN A IS A USEFUL ADDITIONAL HISTOLOGICAL MARKER FOR CUTANEOUS LUPUS ERYTHEMATOSUS

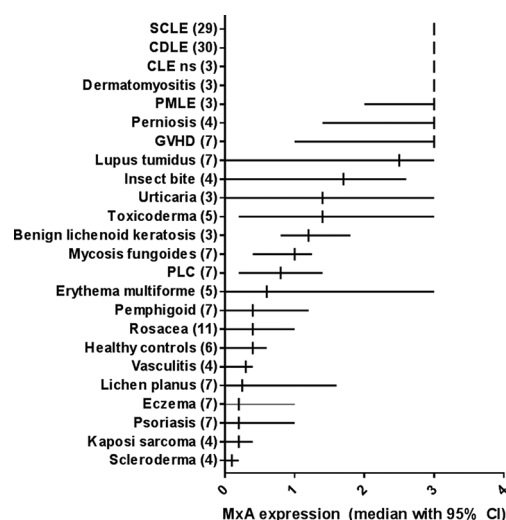
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Background Cutaneous Lupus Erythematosus (CLE) is a heterogeneous auto-inflammatory skin disease, that is to a great extent driven by type I and III interferon (IFN). Histology of skin biopsies plays an important role in the diagnostic confirmation of CLE. Unfortunately, no specific histological marker for CLE is available. In this study, we tested the diagnostic potential of immunostaining with Myxovirus resistance protein A (MxA), which is tightly induced by type I and type III IFN, in CLE skin biopsies.

Methods 178 skin biopsy specimens were collected from the local pathology database. Various skin conditions were selected, provided that clinical diagnosis matched with histological diagnosis. Skin sections were incubated with anti-MxA (R&D systems, AF7946). Consecutively, rabbit anti goat-HRP conjugate (Dako, 0449) was added and sections were stained with diaminobenzidine. The expression of MxA was scored semi-quantitatively.

Results MxA staining was strongly positive in 90.3% of lesional CLE skin sections (except lupus tumidus) and had a negative predictive value of 94%. The same MxA expression pattern was found in dermatomyositis, which is also an IFN-driven autoimmune disease. In some conditions, like perniosis and graft versus host disease, high expression could be found,



Abbreviations: ns = not specified, SCLE = subacute cutaneous lupus erythematosus, CDLE = chronic discoid lupus erythematosus, PMLE = polymorphic light eruption, GVHD = graft versus host disease, PLC = pityriasis lichenoides chronica.

Abstract P27 Figure 1 MxA expression in all analyzed skin diseases (number of biopsies)

but this was less consistent compared to CLE. Most other inflammatory skin diseases did show no or a low expression of MxA. (See figure 1).

Conclusion MxA is strongly expressed in CLE skin with a high negative predictive value and is thus useful as additional diagnostic histological marker, expectedly resulting in restriction of misdiagnosis and treatment delay.

P28 DECREASED PLATELET SIZE IN SYSTEMIC LUPUS ERYTHEMATOSUS IS ASSOCIATED WITH UP-REGULATION OF TYPE I INTERFERON PROTEINS

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Background Dysregulated apoptosis is of major importance in Systemic Lupus Erythematosus (SLE) pathogenesis, linked to the development of autoantibodies, immune complex formation and type I interferon signaling. Platelets from SLE patients are smaller in size, compared to platelets from healthy individuals, which may suggest an increased rate of apoptosis, a known cause of platelet shrinkage. Our aim with this project was to investigate if decreased platelet size could be explained by increased apoptosis rates.

Methods Platelet activation markers; CD62P, CD41, CD154, CD32, PAC-1 and PAR1 and apoptosis; Annexin V, Caspase 3 activation, mitochondrial content (MitoTracker) and mitochondrial depolarization (JC-1) were analyzed in 23 SLE patients and 10 healthy controls (HC) by flow cytometry. Analysis of the total protein content in platelets from SLE patients of normal ($n=5$) and decreased ($n=5$) size were made using mass spectrometry (MS).

Results The level of CD41 ($p=0.001$) positive platelets and mean expression of CD154 ($p=0.004$) were higher in SLE patients. A JC-1 ratio ($p=0.0001$) indicating increased mitochondrial depolarization was significantly associated with

platelets from SLE patients. MS analysis revealed 32 proteins with ≥ 1.5 -fold difference and a p-value of less than 0.05 (Abundance Ratio Adjusted). STAT1, ISG15, NMI and TRIM25 were among 19 proteins expressed at higher levels in small platelets and unbiased enrichments analyses showed a significant overrepresentation of proteins related to type I interferon signaling.

Conclusions The increased mitochondrial depolarization in platelets from SLE patients is an indication but not conclusive evidence of increased platelet apoptosis. Interestingly, decreased sized platelets from SLE patients showed an up regulation of type I interferon related proteins, suggesting direct or indirect influence of IFN. This is a novel finding that may suggest that platelet size is related to IFN signaling. Further studies will be conducted to investigate the mechanistic and potential clinical role of this finding.

P29 CLINICAL RELEVANCE OF DFS70 ANTIBODIES AT A COMMUNITY HOSPITAL

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Background/Purpose Antinuclear antibodies (ANA) are a serological hallmark of systemic autoimmune rheumatic diseases (SARD), such as systemic lupus erythematosus (LES), Sjogren's syndrome, systemic sclerosis and mixed connective tissue disease. Indirect immunofluorescence using human epithelial cells (HEp-2) is the gold standard for ANA screening. The nuclear pattern dense fine speckled (DFS) is one of the most common found when ANA levels are increased. DFS70 antibodies have been detected in inflammatory and neoplastic diseases; in contrast they are rare in SARD. These findings lead to research about their clinical implications. The aims of this study are: understand the meaning of anti-DFS70 antibodies; recognize the relevance of these antibodies in the diagnosis of patients with increased ANA titers in the context of SARD and non-rheumatic pathology (NRP).

Methods A retrospective observational study of consecutive patients observed during 2018 in a community hospital with anti-DFS70 antibodies positivity were performed. Sex, age, clinical and autoantibodies associations were recorded. Ethical approval was obtained and it was executed in compliance with the Helsinki Declaration.

Results Of forty-seven patients 38 (80.9%) were females; mean of age was 51,57 years; 10 (21.3%) were diagnosed with SARD (among which 4 were LES, 5 rheumatoid arthritis and 1 had autoimmune thyroiditis); 10 (21.3%) NRP (2 with asthma, 1 allergic rhinitis, 1 sinusitis, 1 urticaria, 1 vitiligo, 2 neoplasia and 2 Gilbert's syndrome) and 27 (57.4%) had no pathology associated - 4 patients of them presented positive antibodies without SARD.

Conclusions Although DFS70 antibodies are not specific for a particular condition, our study shows that they are a useful biomarker for differentiating between SARD and NRP when others antibodies are presented and it should be considered as a negative predictor for SARD if no other antibody is present. Whereby, DFS70 should be integrated into ANAs initial interpretation algorithm to avoid further studies.

P30 A CUSTOM-MADE MICROARRAY FOR DETECTION OF AUTOANTIBODIES IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Systemic Lupus Erythematosus (SLE) is a heterogeneous autoimmune disease associated with chronic inflammation with progressive organ degeneration. Early diagnosis intervention is crucial to improve overall disease course. Anti-dsDNA antibodies are the most sensitive serological biomarkers for SLE. The available diagnostic methods, however, identify different antibody subpopulations against dsDNA leading to varying performance. Thus, a serological biomarker profile specific to aid timely diagnosis and sensitive for disease activity of SLE remains an important clinical unmet need.

In this study a microarray was developed that enabled a simple and rapid characterization of the serological profile of SLE patients. In this microarray it was possible to analyze up to 90 autoantibodies in $<10 \mu\text{l}$ serum, making it a useful tool for characterization of SLE patient cohorts.

Methods A microarray was developed by spotting autoantigens in an arrayed fashion onto the surface of maxisorp plastic slides. Binding of each antigen was optimized regarding concentration and spotting buffer. The assay was performed by incubating diluted serum on the array followed by incubation with a fluorescent-labeled anti-human IgG antibody. Fluorescence intensities were measured with a laser scanner and the resulting images analyzed with an image analysis software. The microarray performance was compared to single analytes on EliA™ (Phadia AB, Uppsala, Sweden). For technical evaluation serum from 160 SLE patients and 313 healthy blood donors were measured.

Results Overall, there was strong correlations between microarray and EliA results for relevant biomarkers. The mean CV% between productions, lots and assays were 11, 8 and 7%, respectively.

Conclusions The developed microarray showed good technical performance and is a promising tool for the characterization of the serological profile of SLE patients. Furthermore, it has been applied in a clinical study as a discovery tool for new autoantibodies and patterns of autoantibodies with promising results.

P31 ANTI-DOMAIN I POSITIVITY IN SLE AT DIAGNOSIS IS PREDICTIVE OF ATHEROSCLEROTIC PLAQUE DEVELOPMENT

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Background Cardiovascular disease is a significant burden on SLE patients and no satisfactory markers exist to predict atherosclerotic development in patients. This study utilised an orthogonal approach to develop a marker of cardiovascular progression with predictive value in SLE patients.