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 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.

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.

Additional Histological Marker for Cutaneous Lupus Erythematosus

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