AUTOIMMUNE CONNECTIVE TISSUE DISEASES

ORAL LICHEN PLANUS: SALIVARY PROTEOMICS AND ITS RELATIONSHIP WITH PATHOGENESIS

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Introduction: Oral lichen planus (OLP) is a chronic, T-cell-mediated, inflammatory disease that affects the oral cavity. The OLP pathogenesis is still unclear; however, the main evidence is that the mechanisms of activation of different T lymphocytes pathways induce apoptosis with an increase of Th1 and Th17 subtypes cells, triggered by the release of cytokines.

Objective: This study analysed saliva proteomics to identify protein markers that might be involved in the pathogenesis and development of the disease.

Materials and methods: Proteins differentially expressed in the saliva of 10 oral lichen planus and 10 healthy controls were screened using mass spectrometry; the proteins found in OLP were subjected to bioinformatics analysis, including gene ontology and string networks analysis. The multiplex analysis validation allowed the correlation between the proteins identified and the involved cytokines in Th17 response.

Results: All patients showed oral mucosa lichen planus only, without any skin, hair or ungueal involvement, and were diagnosed by histological and immunofluorescence analysis. Our results showed a range of proteins involved in oxidative stress such as Alpha-1-acidic glycoprotein 1 (ORM1), Haemoglobin (HB) and Catalase (CAT). The OLP group, also showed proteins involved with inflammation as albumin (ALB), Zinc-alpha-2-glycoprotein (AZGP1), S100A9, S100A8, Haptoglobin (HP), prolactin-inducible protein (PIP), lysozyme (LYZ), Beta-2-microglobulin (B2M), Heat shock proteins (HSPA5), Thymosin beta-4 (TMSB4). OLP also showed a higher concentration of IL-1β, IL-17F, CD40L and IL-23.

Conclusions: The protein profile demonstrated that the pathogenesis in OLP integrates the chronic inflammatory process which can trigger an increase of oxidative stress, leading to endothelial dysfunction and tissue damage, such as apoptosis and cell proliferation.

Validation of cytokines expression on OLP demonstrated the involvement of S100A8, S100A9, HP and AZGP1 as a trigger of cytokines, that corroborated the association of these proteins and cytokines in a pathological function of this disease and antioxidant activities.