

Disease Activity Report and Inflammatory Conditions in Saliva and Urine

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DESCRIPTION

SLE is a chronic autoimmune illness marked by the activation of both innate and adaptive immunological responses, which can result in serious organ damage. Although the cause of autoimmunity in SLE is unknown, natural immune responses in SLE have been linked to abnormalities in apoptotic clearance, loss of tolerance, and a type I interferon (IFN) profile. Type I IFNs are mainly generated by plasmacytoid dendritic cells in response to immunological components and Neutrophil Extracellular Traps (NETs) formed during neutrophil NETosis. SLE also exhibits systemic inflammation, with elevated levels of pro-inflammatory cytokines that include Tumour Necrosis Factor (TNF)-a, interleukin (IL)-6, and IFN-a, Induced Protein (IP)-10 [1,2].

Monocytes, as well as the transition and activity in response to certain cytokines and chemokines, play an important role in inflammation. Colony-Stimulating Factor (CSF)-1 is the major modulator of mononuclear phagocyte survival, proliferation, differentiation, and function. Notably, CSF-1 encourages monocytes to release high amounts of numerous cytokines, including TNF-, which enhances IP-10 secretion; both appear to be implicated in the aetiology of SLE and associated with renal involvement. Furthermore, CSF-1 has been shown to identify Lupus Nephritis (LN). Monocytes are the primary producers of Monocyte Chemoattractant Protein (MCP)-1. It regulates monocyte migration and infiltration, among other things, and its expression is likewise elevated by TNF- α [3,4].

SLE has a diverse clinical manifestation and can affect numerous organs, including the skin, joints, mucosa, kidneys, neurological system, and bone marrow. The level of impairment and Disease Activity (DA) both influence the degree of severity of SLE, which ranges from moderate to severe and can occasionally result in life-threatening consequences. The mouth and oral mucosa are clearly involved in SLE, and the existence of ulcers in the mouth is included in each of the three major sets of SLE categorization criteria. Another typical oral sign of Secondary Sjögren's Syndrome (SS) is dry mouth [4,5].

Potential DA indicators may be found in bodily fluids other than serum/plasma. Despite the involvement of the oral cavity in

SLE, study of cytokines in saliva as possible illness indicators has received little attention. More urine tests are being conducted, however the results have thus far had limited application in clinical practice. To provide a more full picture, we looked at the amounts of innate immunity-related biomarkers in reflected saliva, serum, and urine samples from SLE patients and community controls in connection to general and renal DA measures. Furthermore, we tested their ability to distinguish SLE patients from controls [6,7].

The potential of the biomarkers investigated to distinguish those suffering from SLE from controls was also evaluated. The AUROC analysis was carried out. TNF-, in particular, was a good a discriminator of SLE patients from controls, with an AUROC of 0.92 in serum. It also performed exceptionally well in the saliva, having an AUROC of 0.85. CSF-1, TNF- α , IP-10, and MCP-1 in both salivary and serum demonstrated the ability to identify SLE patients from control systems, all with AUROC>0.7. Previous observations of TNF- α and CSF-1 in circulating and urine support our findings [8,9].

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