

Lupus: Open Access (LOA)

A Novel Immunoprofiling Technique for Diagnosing SLE and Assessing

Therapy Response

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DESCRIPTION

SLE is a serious autoimmune illness that affects more women than males. Patients with SLE lose immunological tolerance to self-antigens (such as the high-mobility group protein 1) by an unknown mechanism, triggering autoreactive immune responses. Systemic inflammation (which includes lethargy, malar rash, and fever), immunological dysregulation (an elevated levels of autoantibodies as well as low serum complement contents), and damage to organs (such as nephritis, arthritis, and peripheral neuropathy) are common signs of SLE start. Notably, SLE diagnosis is difficult since early-onset SLE symptoms might be non-specific and mirror other more common illnesses [1].

The nature of the variation in immunoprofiling between SLE patients and Healthy Controls (HCs) can describe immune response dysregulation. Patients with SLE exhibit decreased percentages of Natural Killer (NK) cells, Dendritic Cells (DC), regulatory cells, and CD4+/CD8+ T-cell ratios (due to CD4 lymphocytopaenia) and a greater proportion of B cells, double-negative T cells, and regulatory CD4+ T cells when compared to HCs. These changes could be possible targets for assessing the effectiveness of SLE disease treatment [2,3].

Clinical symptoms and serum autoantibodies are used as markers in the traditional SLE diagnosis criteria. The SLE Disease Activity Index (SLEDAI), Systemic Lupus Activity Measure Index, and British Isles Lupus Assessment Group Index (BILAG) are used in clinic to assess disease activity in SLE patients. However, because of the dichotomous and personal evaluation criteria, these measures may not be adequate for monitoring therapy efficacy. Additionally, the lack of a diagnostic index for SLE disease progression based on immunoprofilings limits diagnostic accuracy. As a result, an innovative and precise diagnostic index for SLE supervision is required. Immunoprofilings differ between individuals who have SLE and those with HCs [4,5]. This study aims to analyze the changes in immunoprofilings between SLE patients and healthy controls and to identify the subsets found in SLE. In addition, these characteristics subsets to build a ranking algorithm which helps clinicians in SLE diagnosis. Finally, physicians examined whether

immunoprofilings of SLE patients before and after immunosuppressive treatment are useful for accurate clinical evaluation of disease activity [6,7].

Following that, an immunological signature algorithm was developed based on immunoprofiling from individuals who had SLE and HCs, which demonstrated a high degree of specificity and sensitivity in identifying patients having SLE from HCs.

Monitoring disease activity in SLE is still difficult. The SLEDAI-2K and BILAG-2004 are clinical symptoms and laboratory findings-based methods for assessing disease activity in SLE. However, because its scoring criteria are comparable to those of the 2019 EULAR/ACR criteria, the SLEDAI-2K is unresponsive to symptom improvement. As a result, while research indicates these diagnostic tools might indicate or even predict patient reactions to immunosuppressive medication, an immune cell-based diagnostic tool is currently absent. The immune signature score we developed can provide an exhaustive overview of immune cell dynamics throughout treatment regardless of the existence of clinical symptoms [8-10].

The small sample size was one of the limitations of the study. Larger-scale investigations of the utilization of immunoprofilings in the development of immunological signatures are required for future research. Finally, physicians defined the immunological signatures of SLE patients as well as the dynamics of immunoprofilings following immunosuppressive medication; they may be used as a manifestation of disease activity and hence aid in the diagnosis of SLE.

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