Elevated interleukin-25 and its association to Th2 cytokines in systemic lupus erythematosus with lupus nephritis

ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune disorder that is associated with lupus nephritis, initiated by the deposition of immune complexes in the kidney; subsequently, this induces the overexpression of cytokines. Lupus nephritis is known as one of the major clinical manifestations that affect the disease severity in SLE patients. An increased number of resident periglomerular and immune cells in the kidney has the potential to affect the equilibrium of different immune cell subsets, such as Th1, Th2, Th17, and Tregs, which may be central to the induction of tissue damage in kidney by exerting either proinflammatory or anti-inflammatory effects, or both. This equilibrium has yet to be confirmed, as new players such as IL-25 remain undiscovered. IL-25 is a cytokine of the IL-17 family, which stimulates Th2-mediated immune response when overly expressed. Thus, the aim of this research is to determine the plasma levels of IL-25 and Th2-associated cytokines (IL-4, IL-5, IL-6, IL-9, IL-10, IL-13) in SLE patients with (SLE-LN) and without lupus nephritis. Sixty-four (n = 64) SLE patients and fifteen (n = 15) healthy individuals were recruited. This study demonstrated that the IL-9, IL-10 and IL-25 had significantly increased expressions in SLE-LN, followed by SLE without LN, compared to healthy controls. Meanwhile, IL-5 and IL-6 had significantly reduced. No significant difference was observed with IL-13, while the level of IL-4 was undetectable. Furthermore, IL-9 and IL-10 were significantly correlated with the IL-25, and IL-25, IL-9 and IL-10 were positively correlated with the disease severity score, SLEDAI. In conclusion, IL-25 and its associated Th2 cytokines (IL-9 and IL-10) may be involved in SLE pathogenesis. These cytokines could be potential biomarkers in monitoring and predicting the disease severity during SLE pathogenesis.