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B Cell Repertoire in Lupus Nephritis – Role in Disease Pathogenesis and Implications on Treatment

Desmond Yat-Hin Yap
The University of Hong Kong, Hong Kong

Lupus nephritis (LN) is a serious complication of systemic lupus erythematosus (SLE). The pathogenesis of SLE and LN is highly complex, involving the interaction between different immune-reactive cells, resident renal cells and cytokine pathways. The B cell repertoire plays key pathogenic roles in SLE and LN, including the production of autoantibodies, secretion of pro-inflammatory cytokines and presentation of autoantigens. A plethora of epigenetic regulators, genes and cytokines contribute to the development and maturation of B cells and hence the pathogenesis of SLE and LN. the current standard-of-care induction treatments for severe LN are corticosteroids in combination with cyclophosphamide or mycophenolic acid analogues (MPAA), while maintenance therapies entail the use of low-dose corticosteroids with MPAA or azathioprine. Biologics, calcineurin inhibitors and proliferation signal inhibitors also have emerging roles in the management of LN. These conventional and novel immunosuppressive treatments have improved the clinical outcomes of LN patients and show varying effects on B cells, related cytokines and autoantibodies. The discovery of new biological pathways in B cell and antibody homeostasis may further advance our treatments in autoimmune disorders including SLE and LN. An enhance understanding of B lymphocytes and its biology may propel our knowledge on the pathogenesis of LN, which will potentially improve the disease monitoring and treatment strategies of SLE and LN.