Group 2 innate lymphoid cells suppress pathology of neuromyelitis optica in mice

Ying Kong[1]，Dan Wang[1]，Xiaolin Gao[1]，Chunsheng Yang[1]，Minshu Li[1]，Qiang Liu[1]

[1]Department of Neurology, Tianjin Neurological Institute, Tianjin Medical University General Hospital, Tianjin 300052, China

Abstract

Neuromyelitis optica (NMO) is a severe central nervous system (CNS) autoimmune disease that primarily damages the optic nerves and spinal cord. Group 2 innate lymphoid cells (ILC2) are potent producers of type 2 cytokines that orchestrate immune and inflammatory responses. However, the role of ILC2s in CNS autoimmune disease remains unknown. In NMO patients, we identified a significant reduction of circulating ILC2 in peripheral blood. In a mouse model of NMO induced by intracerebral injection of NMO-IgG and human complement, we found infiltration of ILC2 into the CNS lesions. Notably, a large portion of CNS-infiltrating express IL-5. Depletion of ILC2 led to increased lesion size, astrocyte injury and the loss of aquaporin-4 in the CNS. The exacerbated NMO pathology was accompanied by increased accumulation of microglia/macrophages in the CNS lesions. In addition, expansion of ILC2 using IL-33 attenuates NMO pathology. Collectively, these findings suggest a beneficial role of ILC2 in NMO. Immune interventions targeting ILC2 deserve further investigation as a viable approach to restrict CNS pathology in NMO patients.