عنوان مقاله:

Complete ablation of tumor necrosis factor decreases the production of IgA, IgG, and IgM in experimental central nervous system tuberculosis

محل انتشار:

مجله علوم پایه پزشکی ایران, دوره 23, شماره 5 (سال: 1399)

تعداد صفحات اصل مقاله: 11

نویسندگان:

Ngiambudulu Francisco - Program of Infection and Immunity, the Fifth Affiliated Hospital of Sun Yat-sen University, Zhongshan School of Medicine, Sun Yat-sen University, Guangdong, China|Institute of Tuberculosis Control, Zhongshan School of Medicine, Sun Yat-sen

Nasiema Allie - SAMRC Centre for Tuberculosis Research, Division of Molecular Biology and Human Genetics, Department of Biomedical Sciences, Stellenbosch University, Stellenbosch, South Africa

Boipelo Sebesho - Division of Immunology, Department of Pathology and Institute of Infectious Disease and Molecular Medicine, Faculty of Health Sciences, University of Cape Town, Cape Town Y9Ya, South Africa

Bernhard Ryffel - Institut de Transgenose, CNRS, GEMYPAA, Orleans, France, University of Orleans and CNRS UMRYPAA, Experimental and Molecular Immunology and Neurogenetics, Orleans, France

خلاصه مقاله:

Objective(s): This study aimed to explore the contribution of tumor necrosis factor (TNF) in the recruitment of B-cell and secretion of immunoglobulins (Igs) during cerebral tuberculosis (TB). Materials and Methods: In this work, the contributing role of TNF in regulating Ig secretions was investigated by comparing wild type TNF (TNFf/f), B-cell-derived TNF (BTNF-/-), and complete TNF ablation (TNF-/-) in a mouse cerebral Mycobacterium tuberculosis infection. Using flow cytometry and ELISA, we were able to examine the recruitment of B-cell subsets, and the production of Igs; also assessed the expression of surface markers on B cell subsets. Results: Here, we found that TNF-/- mice showed defective expression of IgA, IgG, and IgM antibodies compared with TNFf/f and BTNF-/- mice, which was significantly decreased in the expression of surface markers and co-stimulatory molecules. Moreover, mice that produced low antibody levels were not able to control infection, therefore progressed to disease; providing direct evidence for the TNF gene-regulating humoral immunity during central nervous system (CNS) M. tuberculosis infection. In contrast, BTNF-/- mice controlled the infection and had levels of IgA, IgG, and IgM comparable to TNFf/f mice.Conclusion: Together, our results demonstrate that TNF may serve as an essential regulator of antibody-mediated immune responses in CNS TB. However, the protective level exhibited by TNF-producing B cells could be defined as baseline protection that could be used in the development of new therapeutic targets or designing new .vaccines

كلمات كليدى:

antibody, Central nervous system, Humoral, Immunity infections, Mycobacterium, Tumor necrosis factor

لینک ثابت مقاله در پایگاه سیویلیکا:

https://civilica.com/doc/1038550

