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Studies on different inflammatory markers in hypo and hyperthyroidism

Kumar Lav Kush Tarun
Magadh University, India

A widespread category of people whose quality of life is affected by a medical condition is the one representing patients with disorders related to thyroid gland function. Described as having multiple causes (from iodine deficiency to post-partum status), thyroid disease affects a significant number of people and it can be seen in any age group. The challenges in front of a patient with thyroid dysfunction (TD) are recording (qualitative or quantitative) alterations of hormonal secretion of the thyroid gland structure, differentiation of psychological effects caused by the manifestation of the disease from the ones caused by specific medication and the importance the patient gives to life events. It is well known that the principle effectors of the immune system are B and T lymphocytes and those lymphocytes can be divided into T-helper (T_h) cells, which express CD4+ surface antigens, and T-cytotoxic (T_c) cells, which express CD8+ surface antigens. The CD4+ T_h precursor cells are further subdivided into two populations, T_{h1} and T_{h2} cells. T_{h1} cells secrete interleukin-2 (IL-2), interferon- ($IFN-$) and tumor necrosis factor alpha (TNF- α), which regulate the cellular-mediated immune response and the induction of tissue damage. T_{h2} cells secrete interleukins, IL-4, IL-5, IL-6 and IL-10 and are activated to provide help to B lymphocytes for specific immunoglobulin (Ig) production. Thyroid autoimmunity has been shown to occur via a two stage process. Stage one involves the increased appearance of intra-thyroid antigen presenting cells (APC) that carry and present thyroid auto-antigens to T_h cells. Stage two involves lymphocytes interacting with the presented autoantigens, leading to the generation of a large number of autoreactive CD4+ T_h lymphocytes, CD8+ T_c lymphocytes and antibody-producing B lymphocytes that infiltrate the thyroid parenchyma. This turns the thyroid gland into a 'battlefield' where the outcome of the interaction between the thyrocytes and infiltrating lymphocytes determines the different clinical outcomes of AITD, believed to be due to differences in the cytokines profile in the thyroid gland upon infiltration. In AIH there appears to be a prevalence of T_{h1} cytokines, leading to a predominance of T lymphocyte immunity, causing increased immune destruction of the thyroid cells and hypothyroidism. In GD, on the other hand, there appears to be a predominance of T_{h2} type inflammatory cytokines and B lymphocyte immunity producing high levels of IgG antibodies specific for the TSHR which can activate the receptor, causing thyroid cell hyperplasia and hyperthyroidism. The fact however, that both diseases can develop in the same individual at different time points suggest that immunological categorizations may be overly simplistic and that cytokine patterns are dynamic processes. This review will focus on the genetic contribution to AITD which to a large extent involves the role of immune response genes.

ltkaran@gmail.com

Specific immune tolerance to spinal antigens as a protective strategy in rats with traumatic spinal cord injury

Lorena Martínez Alcantar, Diana Karina Talavera Carrillo, Ana Laura Pelayo Gómez, Jonathan Uriel Cruz Pineda, Alma Lilia Fuentes Farias and Esperanza Meléndez Herrera
Universidad Michoacana de San Nicolás de Hidalgo, Mexico

Spinal cord injury (SCI) elicits a robust intra-spinal inflammatory response with potentially devastating consequences. Immune cells present in the injury site may sequester cell debris and carry spinal antigens (SAGs) to secondary lymphoid organs. There, SAGs may be processed and presented by antigen presenting cells to lymphocytes, triggering lymphocyte activation. In clinical and experimental SCI, only a few autoantigen targets have been documented. ACAID (anterior chamber associated immune deviation) is an antigen-specific form of peripheral immune tolerance (IT) that is induced against exogenous antigens placed in the anterior chamber (AC) of the eye. It is characterized by the inhibition of delayed hypersensitivity reactions to the AC-injected antigens. This IT is elicited by AC-induced CD4+ CD25+ antigen-specific regulatory T cells. Since neural degeneration after SCI includes a strong inflammatory component triggered by reactivity against multiple antigens derived from the neural tissue, this project proposes to induce IT against SAGs as a neuro-protective strategy in SCI. Results to date indicate that it is possible to induce IT to a cocktail of SAGs obtained from healthy rat spinal cord by ACAID. Neuroprotection of the spinal cord will be evaluated by histological and immuno-fluorescence techniques and further functional evaluations will be done with sensorimotor tests as indicators of spinal cord tissue preservation.

lorenamartinezalcantar@gmail.com