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Subsets of Regulatory T Cells in Sarcoidosis Patients

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Abstract

Many researchers have recently backed the autoimmune theory of sarcoidosis. The presence of an uncontrolled inflammatory response at the local and systemic levels in sarcoidosis patients did not rule out the possibility that immunoregulatory mechanisms were compromised. The purpose of this study was to assess the distribution and disturbance of circulating Treg cell subsets in the peripheral blood of sarcoidosis patients. Materials and procedures: In 2016-2018, a prospective comparative study was conducted. The diagnosis of pulmonary sarcoidosis was made using standard criteria. For Treg immunophenotyping, we used two ten-color antibody combinations.

Keywords: Sarcoidosis • Pathogenesis • Cells • Immunophenotyping

Introduction

Sarcoidosis is a granulomatous disease with a subacute or chronic course, involving the lungs and mediastinal lymph nodes as well as other organs and tissues, with granuloma formation without caseous necrosis. The acute course of the disease has two variants löfgren's heerfordt-waldenström syndrome and löfgren's heerfordt-waldenström syndrome. The formation of granulomas in the lungs, mediastinal lymph nodes, skin, and other organs is a key feature of sarcoidosis pathogenesis. Contact of antigen-presenting cells (macrophages, dendritic cells, epithelial cells) with unknown foreign antigen results in a dysregulated immune response that manifests as granulomatous inflammation in patients who are genetically predisposed to this disease. Sarcoidosis is distinguished by the formation of noncaseating epithelioid granulomas in various organs, which are represented by lymphocytes, epithelioid and giant cells.

In sarcoidosis research, the problem of studying an etiological factor led to the identification of various infectious agents ranging from bacteria to viral agents, fungi, and inorganic factors. As a result, there is a lack of a unified approach to therapy, as well as the ability to conduct preclinical studies on the efficacy of treatment on sarcoidosis models. Many researchers have recently backed the autoimmune theory of sarcoidosis. The aetiology of pulmonary sarcoidosis is still unknown; thus, infections and/or autoimmunity are considered potential triggers of this disease [1].

Literature Review

Because some studies revealed the presence or absence of bacterial and fungal pathogens in the granuloma, other etiologic mechanisms (autoimmunity with the presence of self-reactive T cells and auto-antibodies) were proposed as a possible cause of the disease. The granuloma may be formed by the inflammatory cascade, which includes pro-inflammatory cytokines produced by T-helper cells, macrophages, and monocytes. Recent research suggests that prolonged antigen exposure with endotheliocytes, macrophages, and dendritic cells causes macrophage differentiation and the secretion of proinflammatory cytokines. Antigens were presented to T-lymphocytes, and they differentiated, proliferated, and migrated to the site of inflammation. The accumulation of epithelioid macrophages B and T cells in the inflammatory foci results in the formation of epithelioid granulomas without caseous necrosis.

Discussion

Furthermore, it was discovered that uncontrolled inflammatory responses on both the local and systemic levels in sarcoidosis patients could impair immunoregulatory mechanisms. Tregs are essential in preventing autoimmune aggression. Tregs have a variety of suppression mechanisms that target both immune and non-immune cells, including cell-to-cell contact-dependent suppression, production of anti-inflammatory cytokines, perforin/granzyme-mediated killing of target cells, and so on. Tregs were thought to be able to suppress granuloma development and effector functions of different immune cells in sarcoidosis patients, but an investigation of the total peripheral blood Treg subset in sarcoidosis patients revealed very contradictory data. Several studies have found that sarcoidosis patients have more Tregs than the control group.

Another critical question concerns the cell subset that regulates self-reactive B cell activation as well as self-reactive follicular the activity in germinal centre responses. T follicular regulatory cells have recently been identified in mice and humans. The Tfr cell is thought to play a key role in the specific control of Tfh and B-cell interactions in the germinal centre and to have suppressive abilities, preventing the emergence of a self-reactive clone of B cells. For example, mouse Tfr cells have phenotypic similarities with the cell surface profiles, including cell surface expression, but they also express activated-specific markers [2].

Sarcoidosis is distinguished by the presence of epithelioid non-necrotizing granulomas containing lymphocytes, epithelioid, and giant cells in various organs. The central part of the granuloma contains macrophages, modified macrophages, epithelioid and giant cells with CD4+ T cells, while the peripheral part contains fibroblasts, macrophages and fibrocytes. B lymphocytes are rarely found in granulomas. Furthermore, central necrosis may be detected. It is important to note that the characteristics of lymphocytic infiltration, as well as the overall state of the immune system in sarcoidosis, are similar to those seen in some autoimmune diseases. There was an imbalance of T-helpers and T-follicular helpers towards an increase in the presence of autoimmune processes, impaired memory, and "naive" B cell distribution [3,4].

Defects in Treg cell numbers in circulation, changes in their phenotype, and Tregs with impaired functions at the site of inflammation have been linked to the risk of autoimmune diseases in a variety of animal models and human autoimmune diseases. The current literature on Treg dynamics during sarcoidosis is contentious. Huang et al., for example, reported that Treg levels in bronchoalveolar lavage fluid and peripheral blood samples from sarcoidosis patients decreased. We also noticed a decrease in the absolute number of Treg cells, but no differences in the relative number of Tregs within the total lymphocyte population were found. Treg cells were found in higher numbers in blood samples and BALF from patients with active sarcoidosis [5,6].

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Conclusion

Thus, changes in Treg cell subsets and phenotypes in sarcoidosis patients may be attributed to dysregulated functions at the site of inflammation and granuloma formation. To gain a better understanding of the role of regulatory T cells in sarcoidosis progression and Treg cell subsets, we may identify their cell surface antigens as potential therapeutic targets for sarcoidosis-specific immune therapy. Based on the immune response, our data show that there are more Tregs in the periphery, which could be linked to an imbalance of follicular Th cell subsets and alterations in B cells. respectively. Another limitation is that, while our study focused on the detailed analysis of the Treg phenotype and the identification of different Treg cell subsets, we did not use in vitro functional tests. Furthermore, an analysis of Treg phenotype numbers could fully characterise their functional activity in peripheral inflamed tissues. Finally, while we used fresh peripheral blood samples for Treg cell analysis, the lack of live/dead staining may have been a limitation of our study. Furthermore, the sample size in our study was limited due to patients' availability and willingness to donate large volumes of whole blood for analyses, as well as bronchoalveolar lavage.

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Conflict of Interest

There are no conflicts of interest by author.

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