The significance of GRAVES' disease-related activated monocyte IFN/ SIGLEC1 signaling.

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Introduction

The immune system is dysregulated in Graves' Disease (GD), and immune cell function is abnormal. There has, however, been few prior research on the function of monocytes in the GD pathophysiology. This study sought to determine whether and how monocytes contribute to the pathophysiology of GD. From untreated first GD patients and healthy controls, CD14+ monocytes were extracted. The type I Interferon (IFN) signalling pathway was shown to be among the most elevated signalling pathways in GD monocytes after RNA-seq was used to assess changes in overall mRNA expression in monocytes. In untreated GD patients, type I IFN dramatically elevated Sialic Acid-Binding Immunoglobulin-Like Lectin1 (SIGLEC1) expression, which was linked with thyroid parameters. Following the use of anti-thyroid medications, patient serum SIGLEC1 concentrations decreased. Proinflammatory cytokine and M-CSF expression in monocytes may be inhibited by silencing SIGLEC1 expression. As a result, our investigation provided evidence that type I IFN-mediated monocyte activation might be detrimental to the aetiology of GD. These findings suggested that type I IFN-activated monocytes/macrophages could be inhibited therapeutically to induce GD remission [1].

Loss of immunologic tolerance to self-antigens, inappropriate activation of autoimmune responses, and organ injury are the hallmarks of autoimmune disorders, which are multifactorial complex diseases. Currently, 5-10% of the global population suffers from one of the major causes of sickness and mortality more than 100 different autoimmune disorders. Aside from that, autoimmune disorders have a significant financial impact due to high rates of disability and comorbidity as well as rising medical expenses. However, there is still a lack of knowledge regarding the actual aetiology and pathogenesis of autoimmune disorders. Recent research suggests that the pathophysiology of various autoimmune disorders shares a great deal in common with one another. Consequently, an integrative investigation of several autoimmune illnesses may provide more information regarding the pathophysiology.

Typical autoimmune disorders include Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA), Graves' Disease (GD), and Systemic Sclerosis (SSc). They are similar in that they affect women more frequently than males, that autoantibodies are produced, that immunological abnormalities in CD4+ or CD8+ T cells are triggered, and that there are numerous risk loci for the disorders. Therefore, study on these autoimmune illnesses may reveal common pathophysiological mechanisms [2].

The study of heritable changes in gene function without changes to DNA sequence is referred to as epigenetics. Through epigenetic mechanisms, which provide a vital link between environmental and genetic risk factors for the diseases, a number of environmental risk factors may exert influence over the pathogenesis of autoimmune disorders. One of the main epigenetic mechanisms, DNA methylation is crucial in influencing how genes operate. Recent studies have revealed that dysregulated DNA methylation is a key factor in the development of autoimmune disorders such GD, RA, SLE, and SSc. Therefore, additional research into the DNA methylation patterns associated with these disorders can provide light on their pathophysiology [3].

In the current study, we provide the results of an integrative analysis of the genome-wide DNA methylomes in CD4+ T cells from patients with GD, RA, SLE, and SSc as well as CD8+ T cells from patients with GD and SSc. The disorders listed above are largely caused by autoimmune reactions mediated by CD4+ and CD8+ T lymphocytes. DNA methylation patterns in various autoimmune disorders were shown to be common. Additionally, the potential diagnostic use of certain DNA methylation profiles for certain autoimmune disorders was examined [4,5].

CD14+ Monocyte and Peripheral Blood Mononuclear Cell Isolation

In a nutshell, fresh PBMCs were extracted from GD patients and healthy controls using gradient centrifugation on Ficoll-Paque Plus (GE Healthcare). Following the manufacturer's instructions, CD14+ monocytes were isolated from fresh PBMCs by positive selection magnetic sorting using human CD14 microbeads and LS columns (Miltenyi Biotec, Cologne, Germany). Using flow cytometry, it was determined that more than 95% of the CD14+ monocytes were pure.

References

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