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The Composition of CD4+ T-cells in Patients with Atherosclerosis and its Age-related Characteristics

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Introduction

Atherosclerosis, a chronic inflammatory condition characterized by the accumulation of lipid-laden plaques within arterial walls, is a leading cause of Cardiovascular Diseases (CVDs) such as heart attacks and strokes. The role of immune cells in atherosclerosis is increasingly appreciated, especially that of CD4+ T-cells, which are key players in adaptive immunity. These cells are not merely bystanders in the disease process but actively contribute to both the progression and resolution of atherosclerotic inflammation. Furthermore, the composition and function of CD4+ T-cells exhibit age-related alterations, which are of paramount importance in understanding how atherosclerosis develops and worsens over time. This article explores the composition of CD4+ T-cells in patients with atherosclerosis, emphasizing the role of different subsets and their characteristics, with a particular focus on how these attributes change with aging [1].

CD4+ T-cells play a central role in orchestrating immune responses. Upon activation, these cells differentiate into distinct subsets, each of which performs specific functions. The most prominent CD4+ T-cell subsets relevant to atherosclerosis are Th1, Th2, Th17, and regulatory T-cells (Tregs). Th1 cells are known to produce pro-inflammatory cytokines, such as Interferon-gamma (IFN-γ), which enhance macrophage activation and promote inflammation within atherosclerotic plaques. Th2 cells, on the other hand, produce anti-inflammatory cytokines like Interleukin-4 (IL-4), which counterbalance the Th1 response but may also contribute to tissue remodeling and repair. Th17 cells secrete interleukin-17 (IL-17), driving neutrophil recruitment and amplifying inflammation. Finally, Tregs, which express the transcription factor FoxP3, act as immune modulators, secreting anti-inflammatory cytokines like IL-10 and Transforming Growth Factor-beta (TGF-β) to suppress excessive immune responses and maintain tissue homeostasis [2].

Description

In the context of atherosclerosis, the balance between pro-inflammatory and anti-inflammatory CD4+ T-cell subsets is crucial. A pro-inflammatory bias, often seen in patients with progressive atherosclerosis, is marked by a predominance of Th1 and Th17 cells. These cells exacerbate plaque formation and destabilization, increasing the risk of rupture and subsequent cardiovascular events. Conversely, an adequate presence of Tregs has been shown to confer protection against atherosclerosis by limiting immune-mediated tissue damage and promoting plaque stability. However, in atherosclerosis patients, especially the elderly, this balance is frequently disrupted [3].

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Age-related changes in the immune system, collectively known as immunosenescence, significantly influence the composition and function of CD4+ T-cells in atherosclerosis. With age, there is a general decline in immune competence, characterized by reduced naïve T-cell production and an expansion of memory T-cell subsets. This shift is associated with chronic low-grade inflammation, or "inflammaging," a hallmark of aging that contributes to the development and progression of atherosclerosis. As individuals age, the thymus—the primary organ responsible for T-cell development—undergoes involution, leading to a reduced output of naïve CD4+ T-cells. In turn, the immune system becomes increasingly dependent on memory T-cells, which have a more limited repertoire for responding to new antigens and are more prone to exerting pro-inflammatory effects [4].

One of the key age-related changes in CD4+ T-cells in atherosclerosis is the altered ratio of effector to regulatory T-cell subsets. In elderly patients, there is often an expansion of pro-inflammatory CD4+ T-cells, particularly Th1 and Th17 cells. These cells contribute to the chronic inflammation seen in aging and atherosclerosis by secreting higher levels of cytokines like IFN-γ, IL-17, and Tumor Necrosis Factor-alpha (TNF-α). This inflammatory environment not only accelerates the progression of atherosclerotic plaques but also leads to their destabilization, making older individuals more susceptible to adverse cardiovascular events. Additionally, aging is associated with a decline in Treg numbers and function, weakening the immune system's ability to control excessive inflammation and prevent immune-mediated damage within the vascular system [5].

Conclusion

In conclusion, CD4+ T-cells play a crucial role in the pathogenesis of atherosclerosis, with different subsets exerting either pro-inflammatory or anti-inflammatory effects. The balance between these subsets is critical for determining the progression and outcome of the disease. In elderly individuals, age-related changes in the composition and function of CD4+ T-cells exacerbate the inflammatory response and promote the development of more severe forms of atherosclerosis. The expansion of pro-inflammatory Th1 and Th17 cells, coupled with the decline in Treg function and TCR diversity, contributes to the chronic inflammation and immune dysregulation observed in aging. These age-related alterations in CD4+ T-cell composition have significant clinical implications, as they increase the risk of adverse cardiovascular events and complicate the management of atherosclerosis in elderly patients. Understanding the age-related characteristics of CD4+ T-cells in atherosclerosis is essential for developing targeted therapies that can modulate immune responses and improve outcomes in this vulnerable population.

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Conflict of Interest

None.

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