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Commentary

The Role of ThGM Cells as a Marker for Predicting Coronary Heart Disease

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DESCRIPTION

A range of immune cells and inflammatory mediators are involved in the chronic inflammatory illness known as Coronary Heart Disease (CHD). The origin of CHD is unknown, and its pathophysiology is complex and varied. To maximize the advantages of medical intervention, early-stage CHD must be detected. It has been suggested that CD4+T cells, macrophages, neutrophils, high-sensitivity C-reactive protein, IL-6, and GM-CSF play a part in the onset and progression of CHD. The condition necessary for CHD is atherosclerosis. Immune cells from the blood, such as mast cells, macrophages, and T lymphoid cells, make up the majority of atherosclerotic plaques.

T lymphoid cells among them are significant. In the underlying atherosclerotic lesions, T helper (Th)1 and T helper (Th)17 cells had an active pro-inflammatory state. Interferon-gamma (IFN-), a potent pro-inflammatory cytokine that is mostly produced by Th1 cells, has been found in atherosclerotic plaques in both people and animals. Th17 cells produce IL-17 and, to a lesser extent, TNF- and Interleukin-6, which are both essential for the growth of atherosclerotic lesions. Although its mechanism is still unknown, it has recently become widely accepted that Th cells (ThGM) that produce GM-CSF play a role in the onset and development of various autoimmune disorders. The IL-7-STAT5 pathway aided in the promotion of GM-CSF expression in ThGM. GM-CSF are pro-inflammatory cytokines that participate in the pathogenesis of many kinds of autoimmune diseases, such as Experimental Autoimmune Encephalomyelitis (EAE), Multiple Sclerosis (MS), and Myasthenia Gravis (MG). In MG patients, the frequency of GM-CSF-producing T cells was decreased in the blood, but enriched in the inflamed thymus. Additionally, the frequency of T cells that produce GM-CSF in the thymus and the severity of MG patients' clinical condition were associated.

Although its mechanism is still unknown, it has recently become widely accepted that Th cells (ThGM) that produce GM-CSF play a role in the onset and development of various autoimmune disorders. The IL-7-STAT5 pathway aided in the promotion of GM-CSF expression in ThGM. Multiple Sclerosis (MS), Experimental Autoimmune Encephalomyelitis (EAE), and myasthenia gravis are among the autoimmune illnesses that are influenced by pro-inflammatory cytokines called GM-CSF (MG). In MG patients, the frequency of GM-CSF-producing T cells was decreased in the blood, but enriched in the inflamed thymus. Additionally, the frequency of T cells that produce GM-CSF in the thymus and the severity of MG patients' clinical condition were associated. Patients with CHD have more ThGM cells, but there is no rise in plasmatic GM-CSF.

These findings suggested that ThGM cells may be dormant in CHD patients. Anti-CD3 and anti-CD28 antibodies may stimulate the growth of ThGM cells *in vitro*. More complicated *in vivo* pathways control ThGM proliferation and differentiation. In ThGM cells, the IL-7-STAT5 pathway stimulates the transcription of GM-CSF. When certain conditions are met, ThGM cells release GM-CSF into the plasma. To keep the concentration of GM-CSF in plasma constant, this process needs to be accurately controlled. Patients with SAP and UAP exhibit higher CD3+IFN+ cell proportions. Additionally, CHD patients' peripheral blood showed an unexpected rise in the number of Th1 cells. We discovered that CHD patients had considerably more Th1 cells (CD4 IFN-+ T cells). It is important to note that the proportion of Th1 cells in AMI patients is normal. According to these findings, Th1 cells are only involved in the initial stages of atheromatous plaque development. ThGM was a recently discovered Th subgroup, and there was a direct correlation between the amount of ThGM cells and the severity of CHD.

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