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Hypertension and Inflammation in Atherosclerosis

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Introduction

The growing body of evidence suggests that inflammation in the vessels plays a significant part in the development of atherosclerosis. It is believed that the initiation and progression of atherosclerosis are dependent on both innate and adaptive immune responses, which typically consist of monocytes, macrophages, neutrophils, T lymphocytes, and B lymphocytes. In addition, traditional low-density or high-density lipoprotein cholesterol and inflammatory biomarkers like high-sensitivity C-reactive protein and interleukin-6 are known to predict future cardiovascular events. We are looking into new therapeutic options that could reduce the rate of critical cardiovascular events by reducing vascular inflammation because of our current understanding of the inflammatory mechanisms of atherosclerosis.

Among the clinically significant cardiovascular diseases (CVDs), atherosclerosis is a progressive pathology that causes coronary artery disease, stroke, and peripheral arterial disease. Since the discoveries made by Rudolf Virchow in the 1850s, it has been widely accepted that atherosclerosis is a chronic inflammatory disease caused by the accumulation of fat in the artery wall and vascular injury. It is common knowledge that both innate and adaptive immune responses play important roles in the beginning and progression of atherosclerosis, ultimately leading to clinical symptoms of cardiovascular disease (CVD), according to numerous studies. The possibility of reducing cardiovascular events and risks by treating inflammation itself has emerged as a result of our current comprehension of the inflammatory pathways that are involved in atherosclerosis. Canakinumab, a therapeutic monoclonal antibody that targets interleukin (IL)-1, significantly reduced recurrent cardiovascular events in patients with stable coronary artery disease at high inflammatory risk, as reported in the Canakinumab Anti-inflammatory Thrombosis Outcomes Study (CANTOS) trial [1].

Description

Atherosclerosis is caused by endothelial damage or the accumulation of oxidized or altered low-density lipoproteins (LDLs) in the artery wall, according to a number of studies. These altered or oxidized LDLs cause both innate and adaptive immune responses, as well as low-grade inflammation caused by endothelial damage. It is now believed that these immunological responses play a significant role in the progression of atherosclerosis [2]. Monocytes/macrophages, neutrophils, T lymphocytes, and B lymphocytes are the primary cell subtypes in atherosclerosis.

Endothelial damage characterized by low-grade inflammation is what causes upregulation of cell adhesion molecules like selectin, vascular cell adhesion molecule-1 (VCAM-1), and intercellular adhesion molecule-1 (ICAM-1). These chemicals help monocytes stick to endothelial cells. Monocytes migrate under the endothelium after attaching to damaged endothelial cells, and several chemokines have been linked to this process. Monocyte chemoattractant protein-1 (MCP-1) encourages monocyte migration and infiltration via its C-C chemokine receptor 2 receptor. Through the leukocyte-specific C-X-C chemokine receptor type 2 (CXCR2), IL-8 and fractalkine are also linked to cell migration. A crucial stage in the progression of atherosclerosis is when monocytes migrate into the endothelium, where they are transformed into macrophages by macrophage colony-stimulating factor (M-CSF) [3].

Scavenger receptors on macrophages enable them to absorb oxidized LDLs. The scavenger receptor family (SR-B1) includes the scavenger receptor class A (SR-A), cluster of differentiation (CD) 36, lectin-like oxidized LDL receptor-1 (LOX-1), scavenger receptor for phosphatidylserine and oxidized lipoprotein (SR-PSOX), and scavenger receptor class B type 1. These receptors enable macrophages to absorb oxidized LDL, resulting in lipid accumulation and the production of foam cells. Atherosclerosis has also been linked to macrophage-expressed toll-like receptors (TLRs), which are known to play a significant role in innate immunity. Oxidized LDLs are thought to activate TLR signaling, escalating plaque inflammation.

Based on the premise that chronic inflammation contributes to the etiology of atherosclerosis, numerous studies have reported that several biomarkers of inflammation could predict future cardiovascular events in patients with CVDs as well as those who appear to be healthy. There is growing evidence that CRP measured by a high-sensitivity assay (hsCRP) can predict future cardiovascular events independently of established risk factors, despite the fact that CRP levels rise in response to a variety of non-specific inflammatory stimuli. In addition, it has been reported that hsCRP poses at least the same cardiovascular risk as established risk factors like hypertension or hyperlipidemia [4].

hsCRP has emerged as a crucial biomarker for predicting cardiovascular risk on the basis of these findings. Indeed, hsCRP is a useful cardiovascular risk assessment tool. It has been demonstrated that the Reynolds risk score's established risk factors, such as hsCRP and family history, can better predict future risks overall. Research into whether treating chronic inflammation could halt the progression of atherosclerosis and, as a result, reduce the number of cardiovascular events has been prompted by the idea and processes of inflammation as a contributor to atherosclerosis. However, it should be noted that inflammation is a component of a pathway that, when traditional risk factors are present, contributes to atherosclerosis and its consequences. Aspirin's cardioprotective effects are thought to be due to its antiplatelet properties rather than its direct anti-inflammatory properties, which is true. Therefore, idea validation should be handled with caution [5]. Researchers require an intervention that reduces inflammation without significantly affecting other atherothrombosis pathways and has a safety profile that permits testing in clinical trials in order to test the inflammatory hypothesis of atherosclerosis.

Conclusion

After a large body of research demonstrating that lowering inflammation may be a promising new method for reducing atherosclerosis, the CANTOS study strongly confirmed the hypothesis that inflammation is directly involved in the etiology of atherosclerosis. A new treatment strategy for reducing cardiovascular disease (CVD) is emerging in clinical practice by focusing on inflammation as the cause of atherosclerosis. On the other hand, anti-inflammatory treatments must be used with caution because they may worsen or cause negative outcomes like infection. In fact, the CANTOS study found that canakinumab was more likely than placebo to cause fatal infections and

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sepsis. Furthermore, more research is required to determine which anti-inflammatory treatments are most effective in preventing atherosclerosis because IL-1 is only one of many potential therapeutic targets for inflammation.

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