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Inflammation in Atherosclerosis

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Introduction

Vascular inflammation appears to have a key role in the pathogenesis of atherosclerosis, according to mounting data. Both innate and adaptive immune responses, which mostly consist of monocytes, macrophages, neutrophils, T lymphocytes, and B lymphocytes, are thought to be critical for the initiation and progression of atherosclerosis. Furthermore, inflammatory biomarkers such as high-sensitivity C-reactive protein and interleukin-6, as well as traditional low-density or high-density lipoprotein cholesterol, are known to predict future cardiovascular events. As a result of our present understanding of the inflammatory mechanisms of atherosclerosis, we're looking at new therapeutic options that could reduce the rate of critical cardiovascular events by reducing vascular inflammation.

Atherosclerosis is a progressive pathology that leads to a variety of clinically significant cardiovascular illnesses (CVDs), including coronary artery disease, stroke, and peripheral arterial disease. Since Rudolf Virchow's discoveries in the 1850s, it has been widely accepted that atherosclerosis is a chronic inflammatory disease that occurs in response to vascular injury as well as the accumulation of fat within the artery wall. Numerous studies have revealed the molecular underpinnings of inflammation in atherosclerosis, and it is commonly known that both innate and adaptive immune responses play important roles in the beginning and progression of atherosclerosis, ultimately leading to CVD clinical symptoms. The current understanding of the inflammatory pathways of atherosclerosis has led to the possibility of reducing cardiovascular events and risks by treating inflammation itself. The Canakinumab Anti-inflammatory Thrombosis Outcomes Study (CANTOS) trial recently reported that using canakinumab, a therapeutic monoclonal antibody targeting interleukin (IL)-1, patients with stable coronary artery disease at high inflammatory risk had a significant reduction in recurrent cardiovascular events [1].

Description

Atherosclerosis pathophysiology

A number of studies have found that endothelial damage or the buildup of low-density lipoproteins (LDLs) within the artery wall, which are prone to oxidation or alteration, causes atherosclerosis. Both innate and adaptive immunological responses are triggered by these changed or oxidized LDLs, as well as low-grade inflammation induced by minor endothelial damage. These immunological responses are now thought to have a key role in the progression of atherosclerosis [2]. In the context of atherosclerosis, the main cell subtypes include monocytes/macrophages, neutrophils, T lymphocytes, and B lymphocytes.

Upregulation of cell adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1), and selectin is triggered by endothelial damage characterized by low-grade inflammation. Monocytes use these chemicals to adhere to endothelial cells. After attaching to wounded endothelial cells, monocytes migrate under the endothelium, and several chemokines have been linked to this process. Through its receptor C-C chemokine receptor 2, monocyte chemoattractant protein-1 (MCP-1) stimulates monocyte migration and infiltration (CCR2). IL-8 and fractalkine are also linked to cell migration via the C-X-C chemokine receptor type 2 (CXCR2), which is found in leukocytes. Monocytes migrate into endothelium, where they are differentiated into macrophages by macrophage colony-stimulating factor (M-CSF), which is a critical phase in the development of atherosclerosis [3].

To take up oxidized LDLs, macrophages have scavenger receptors. 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 are all members of the scavenger receptor family (SR-B1). These receptors allow oxidized LDLs to be taken up by macrophages, resulting in lipid buildup and the production of foam cells. Furthermore, macrophage-expressed toll-like receptors (TLRs), which are known to play a major role in innate immunity, have been linked to atherosclerosis. TLR signaling is thought to be activated by oxidized LDLs, causing plaque inflammation to worsen.

Numerous studies have reported that several biomarkers of inflammation could predict future cardiovascular events in not only patients with CVDs but also in apparently healthy persons, based on the premise that chronic inflammation contributes to the etiology of atherosclerosis. Despite the fact that CRP levels rise in response to a variety of non-specific inflammatory stimuli, there is growing evidence that CRP assessed by a high-sensitivity assay (hsCRP) can predict future cardiovascular events independently of established risk factors. Furthermore, the cardiovascular risk associated with hsCRP has been reported to be at least as high as that associated with established risk factors such as hyperlipidemia or hypertension [4].

Based on these findings, hsCRP has emerged as a key biomarker for predicting cardiovascular risk. Indeed, hsCRP is an effective measure for assessing cardiovascular risk. The addition of hsCRP, along with family history, to established risk factors in the Reynolds risk score, for example, has been shown to increase overall future risk prediction. The notion and processes of inflammation as a contributor to atherosclerosis have prompted research into whether addressing chronic inflammation could prevent atherosclerosis progression and, as a result, minimize cardiovascular events. It should be noted, however, that inflammation is part of a pathway that contributes to atherosclerosis and its consequences when classic risk factors are present. Indeed, the classic anti-inflammatory medicine aspirin's cardioprotective effects are thought to be attributable to its antiplatelet qualities rather than its direct anti-inflammatory actions. As a result, idea validation should be carried out and handled with caution [5]. To test the inflammatory hypothesis of atherosclerosis, researchers need an intervention that reduces inflammation without having a major influence on other atherothrombosis pathways, as well as a safety profile that allows for testing in clinical trials.

Conclusion

The CANTOS study strongly verified the hypothesis that inflammation

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is directly implicated in the etiology of atherosclerosis, after a vast body of research showing that lowering inflammation may be a promising novel method for reducing atherosclerosis. In clinical practice, targeting inflammation as a cause of atherosclerosis is becoming a new treatment approach for lowering CVDs. Anti-inflammatory methods, on the other hand, must be used with caution because they may exacerbate or trigger negative outcomes such as infection. In fact, the CANTOS trial found that canakinumab was associated with a greater rate of fatal infection and sepsis than placebo. Furthermore, because IL-1 is just one of many potential therapeutic targets for inflammation, more research is needed to determine which anti-inflammatory treatments are most viable or applicable for atherosclerosis prevention.

Conflict of Interest

The author declares that there is no conflict of interest associated with this manuscript.

References

1. Ridker, Paul M., Brendan M. Everett, Tom Thuren, and Jean G. MacFadyen, et al. "Antiinflammatory therapy with canakinumab for atherosclerotic disease." *N Engl J Med* 377 (2017): 1119-1131.
2. Libby, Peter, Paul M. Ridker, Göran K. Hansson, and Leducq Transatlantic Network on Atherothrombosis. "Inflammation in atherosclerosis: from pathophysiology to practice." *J Am Coll Cardiol* 54 (2009): 2129-2138.
3. Sage, Andrew P., and Ziad Mallat. "Multiple potential roles for B cells in atherosclerosis." *Ann Med* 46 (2014): 297-303.
4. Ridker, Paul M. "From C-reactive protein to interleukin-6 to interleukin-1: Moving upstream to identify novel targets for atheroprotection." *Circulation Res* 118 (2016): 145-156.
5. Libby, Peter, Paul M. Ridker, and Attilio Maseri. "Inflammation and atherosclerosis." *Circulation* 105 (2002): 1135-1143.

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