Atherosclerotic Plagues and Immune Microenvironment: From Lipid-Lowering to Anti-inflammatory and Immunomodulatory Drug Approaches in Cardiovascular Diseases

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Abstract : A growing number of studies indicate that atherosclerotic coronary artery disease (CAD) has a complex pathogenesis that extends beyond cholesterol intimal infiltration. The atherosclerosis process may involve an immune microenvironmental condition driven by local activation of the adaptive and innate immunity arrays, resulting in the formation of atherosclerotic plaques. Therefore, despite the wide usage of lipid-lowering agents, these devastating coronary diseases are not averted either at primary or secondary prevention levels. Many trials have recently shown an interest in the immune targeting of the inflammatory process of atherosclerotic plaques, with the promised improvement in atherosclerotic cardiovascular disease outcomes. This recently includes the immune-modulatory drug "Canakinumab" as an anti-interleukin-1 beta monoclonal antibody in addition to "Colchicine," which's established as a broad-effect drug in the management of other inflammatory conditions. Recent trials and studies highlight the importance of inflammation and immune reactions in the pathogenesis of atherosclerosis and plaque formation. This provides an insight to discuss and extend the therapies from old lipid-lowering drugs (statins) to anti-inflammatory drugs (colchicine) and new targeted immune-modulatory therapies like inhibitors of IL-1 beta (canakinumab) currently under investigation.

Keywords : atherosclerotic plagues, immune microenvironment, lipid-lowering agents, and immunomodulatory drugs **Conference Title :** ICCCS 2024 : International Conference on Cardiology and Cardiac Surgery **Conference Location :** Berlin, Germany

Conference Dates : July 22-23, 2024

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