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Opinion Article

Inflammation in Atherosclerosis: From Mechanistic Insights to Therapeutic Targeting

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DESCRIPTION

Atherosclerosis, long recognized as the pathological substrate underlying most cardiovascular diseases, has undergone a conceptual transformation over the past several decades. Once viewed primarily as a passive lipid storage disease characterized by cholesterol deposition in the arterial wall, atherosclerosis is now understood as a complex, dynamic inflammatory process involving interactions between lipoproteins, vascular wall components, and numerous immune cell populations. This evolving understanding has expanded our therapeutic horizons beyond traditional lipid-lowering strategies to encompass novel anti-inflammatory approaches, representing one of the most significant paradigm shifts in contemporary cardiovascular medicine.

The "response to injury" hypothesis, first proposed by Russell Ross in the 1970s, laid the groundwork for our current understanding of atherosclerosis as an inflammatory disease. According to this model, endothelial dysfunction represents the initiating event, triggered by various insults including dyslipidemia, hypertension, smoking, and other cardiovascular risk factors. The resulting endothelial activation promotes adhesion and transmigration of leukocytes, particularly monocytes, into the subendothelial space. Within the intima, monocytes differentiate into macrophages that engulf modified lipoproteins, primarily oxidized Low-Density Lipoprotein (LDL), forming characteristic foam cells. This inflammatory cascade is perpetuated by the release of cytokines, chemokines, and growth factors from activated immune cells and vascular cells, ultimately leading to smooth muscle cell proliferation, extracellular matrix production, and the development of complex atherosclerotic plaques.

The role of inflammation extends beyond plaque formation to influence plaque progression and, critically, plaque destabilization. Vulnerable plaques-those prone to rupture and thrombosis-typically exhibit thin fibrous caps, large necrotic cores, and abundant inflammatory infiltrates, particularly macrophages expressing matrix metalloproteinases and other proteolytic enzymes that weaken the fibrous cap. T lymphocytes, particularly T-helper 1 (Th1) cells producing interferon-γ, further contribute to plaque vulnerability by inhibiting collagen synthesis by smooth muscle cells and promoting macrophage activation. Additionally, inflammatory mediators can trigger endothelial erosion, another mechanism of acute thrombotic complications.

The integration of multiple biomarkers representing different pathophysiological pathways, termed "multi-omic" approaches, represents an emerging frontier in precision hypertension management. Proteomic analyses have identified protein signatures associated with incident hypertension and cardiovascular outcomes, while metabolomic profiling has revealed distinctive metabolite patterns in resistant hypertension and secondary forms such as primary aldosteronism. The combination of genomic, proteomic, metabolomic, and traditional clinical data through advanced computational methods may eventually enable more precise patient stratification and treatment selection.

Digital health technologies are increasingly recognized as enablers of precision hypertension management. Wearable and home-based blood pressure monitoring devices provide rich, longitudinal data on blood pressure patterns, including circadian variations, response to medications, and associations with lifestyle factors such as physical activity, diet, and stress. Machine learning algorithms applied to these dense datasets can potentially identify individual-specific blood pressure determinants and predict responses to various therapeutic interventions, guiding more personalized management strategies.

CONCLUSION

The future of hypertension management likely involves a hybrid approach that combines elements of precision medicine with population-based strategies. While individualized therapy selection based on genetic and phenotypic characteristics may optimize outcomes for some patients, particularly those with resistant or complicated hypertension, simplification and

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standardization of treatment protocols will remain important for improving population-level blood pressure control, especially in resource-limited settings. The challenge lies in determining which patients are most likely to benefit from more intensive phenotyping and personalized approaches versus those who can be effectively managed with standard algorithms.