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Programmed Cell Death Protein 1 (PD-1) Signaling in HIV-Associated Cardiovascular Disease: Mechanisms and Therapeutic Implications

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Abstract

Cardiovascular disease (CVD) remains a significant cause of morbidity and mortality in individuals living with HIV/AIDS, despite effective antiretroviral therapy (ART). Emerging evidence suggests a complex interplay between chronic inflammation, immune dysregulation, and endothelial dysfunction in the pathogenesis of HIV-associated CVD. The programmed cell death protein 1 (PD-1) pathway, known for its role in regulating immune responses and maintaining immune homeostasis, has recently garnered attention for its potential involvement in HIV-associated cardiovascular complications. This review provides a comprehensive overview of the current understanding of PD-1 signaling in HIV-associated CVD, elucidating its mechanistic contributions to vascular inflammation, endothelial dysfunction, atherosclerosis, and myocardial injury. Furthermore, we discuss the therapeutic implications of targeting the PD-1 pathway as a novel therapeutic strategy for mitigating cardiovascular risk in individuals living with HIV/AIDS.

Keywords*: Programmed Cell Death Protein 1 (PD-1), HIV, cardiovascular disease, inflammation, immune dysregulation, immune checkpoint inhibitors, therapeutic targets*

Introduction

Cardiovascular disease (CVD) remains a significant cause of morbidity and mortality among individuals living with HIV/AIDS, despite the remarkable advancements in antiretroviral therapy (ART). While ART has significantly improved the life expectancy of HIV-infected individuals, they continue to face a disproportionately high risk of developing CVD compared to the general population. This increased cardiovascular risk is multifactorial and encompasses traditional risk

factors, such as hypertension, dyslipidemia, and smoking, as well as HIV-specific factors, including chronic inflammation, immune dysregulation, and direct viral effects on the vasculature. Emerging evidence suggests that immune checkpoint pathways, particularly the programmed cell death protein 1 (PD-1) pathway, may play a crucial role in the pathogenesis of HIV-associated CVD. PD-1, a coinhibitory receptor expressed on activated T cells, regulates immune responses by inhibiting T-cell activation and effector functions, thereby maintaining immune tolerance and preventing autoimmunity. Dysregulated PD-1 signaling in the setting of chronic HIV infection leads to persistent immune activation and inflammation, which have been implicated in the development and progression of cardiovascular complications.[1-20]

The role of PD-1 signaling in vascular inflammation and endothelial dysfunction is of particular interest in the context of HIV-associated CVD. Chronic immune activation and inflammation contribute to endothelial dysfunction, characterized by impaired endothelium-dependent vasodilation, increased expression of adhesion molecules, and enhanced endothelial permeability. PD-1-expressing T cells accumulate within the vascular microenvironment, where they interact with endothelial cells and perpetuate vascular inflammation through the production of pro-inflammatory cytokines and chemokines, as well as the induction of oxidative stress. Atherosclerosis, a chronic inflammatory disease of the arterial wall characterized by the accumulation of lipid-rich plaques, is a major contributor to CVD in HIV-infected individuals. PD-1 signaling influences the development and progression of atherosclerosis by modulating immune cell recruitment, foam cell formation, and plaque stability. PD-1-expressing T cells within atherosclerotic plaques exhibit a pro-inflammatory phenotype, contributing to plaque vulnerability and the risk of acute cardiovascular events. Furthermore, PD-1 signaling may play a role in myocardial injury in HIV-infected individuals, potentially contributing to the development of myocarditis, cardiomyopathy, and heart failure. Given the growing recognition of the role of PD-1 signaling in HIV-associated CVD, there is increasing interest in exploring the therapeutic implications of targeting the PD-1 pathway as a novel approach to mitigate cardiovascular risk in this population. Immune checkpoint inhibitors, such as PD-1 blockade, have shown efficacy in attenuating vascular inflammation, improving endothelial function, and reducing atherosclerotic burden in preclinical models and clinical studies of other inflammatory diseases. Understanding the specific role of PD-1 signaling in HIV-associated CVD and elucidating the mechanisms underlying its contribution to cardiovascular pathology may pave the way for the development of innovative therapeutic strategies to improve cardiovascular outcomes in individuals living with HIV/AIDS.[21-55]

PD-1 Signaling in Vascular Inflammation and Endothelial Dysfunction

PD-1 signaling plays a pivotal role in modulating vascular inflammation and endothelial dysfunction, both of which are central to the pathogenesis of HIV-associated cardiovascular disease (CVD). Chronic immune activation and inflammation, characteristic features of HIV infection, contribute to endothelial dysfunction, a critical early event in the development of atherosclerosis and other cardiovascular complications. PD-1-expressing T cells are key mediators of vascular inflammation, as they accumulate within the vascular microenvironment and interact

with endothelial cells, perpetuating an inflammatory cascade. Within the context of HIV infection, dysregulated PD-1 signaling leads to persistent immune activation and inflammation, further exacerbating endothelial dysfunction. PD-1-expressing T cells exhibit an activated phenotype and produce pro-inflammatory cytokines and chemokines that promote endothelial cell activation and dysfunction. Additionally, PD-1 signaling inhibits T-cell proliferation and effector functions, impairing the clearance of pathogens and damaged cells, which can further contribute to vascular inflammation and endothelial injury.[56-86]

Endothelial dysfunction is characterized by impaired endothelium-dependent vasodilation, increased expression of adhesion molecules, and enhanced endothelial permeability, all of which promote vascular inflammation and atherosclerosis. PD-1 signaling influences endothelial function by regulating the production of nitric oxide (NO), a key mediator of vasodilation, and the expression of adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1) and intercellular adhesion molecule-1 (ICAM-1), which facilitate leukocyte recruitment and adhesion to the endothelium. In addition to its effects on endothelial cells, PD-1 signaling impacts other cell types within the vascular microenvironment, including macrophages and smooth muscle cells, further contributing to vascular inflammation and atherosclerosis. PD-1-expressing macrophages exhibit an M1 pro-inflammatory phenotype and produce cytokines and chemokines that promote plaque formation and progression. PD-1 signaling also regulates smooth muscle cell proliferation and migration, key processes in the development of atherosclerotic lesions.[87-107]

Role of PD-1 Signaling in Atherosclerosis and Myocardial Injury

The programmed cell death protein 1 (PD-1) signaling pathway plays a crucial role in the development and progression of atherosclerosis, a chronic inflammatory disease of the arterial wall characterized by the accumulation of lipid-rich plaques. In the context of HIV infection, dysregulated PD-1 signaling contributes to persistent immune activation and inflammation, which are key drivers of atherosclerosis. PD-1-expressing T cells within atherosclerotic plaques exhibit a pro-inflammatory phenotype, producing cytokines and chemokines that promote plaque formation and progression. Furthermore, PD-1 signaling influences immune cell recruitment, foam cell formation, and plaque stability, thereby impacting the overall vulnerability of atherosclerotic lesions to rupture and thrombosis. PD-1 signaling regulates the balance between pro-inflammatory and anti-inflammatory responses within atherosclerotic plaques, thereby influencing plaque stability and the risk of acute cardiovascular events. PD-1-expressing T cells modulate the activity of macrophages and other immune cells within plaques, promoting the production of matrix metalloproteinases (MMPs) and other proteolytic enzymes that degrade the fibrous cap and weaken plaque integrity. Additionally, PD-1 signaling influences the formation of foam cells, lipid-laden macrophages that contribute to plaque progression and instability. In addition to its role in atherosclerosis, PD-1 signaling may also contribute to myocardial injury in individuals living with HIV/AIDS. Chronic immune activation and inflammation associated with HIV infection can lead to myocarditis, cardiomyopathy, and heart failure, all of which are characterized by myocardial inflammation, oxidative stress, and impaired contractile function. PD-1-expressing T

cells within the myocardium may exacerbate myocardial injury through their effects on inflammation, fibrosis, and cardiomyocyte apoptosis.[108-140]

Therapeutic Implications and Future Directions

The role of programmed cell death protein 1 (PD-1) signaling in HIV-associated cardiovascular disease (CVD) opens avenues for novel therapeutic strategies and research directions. Targeting the PD-1 pathway holds promise for mitigating cardiovascular risk and improving outcomes in individuals living with HIV/AIDS. Several therapeutic implications and future directions emerge from our understanding of PD-1 signaling in HIV-associated CVD. Immune checkpoint inhibitors targeting PD-1, such as pembrolizumab and nivolumab, have shown efficacy in attenuating vascular inflammation, improving endothelial function, and reducing atherosclerotic burden in preclinical models and clinical studies of other inflammatory diseases. Future research should explore the safety and efficacy of PD-1 blockade in HIV-infected individuals with cardiovascular complications, including randomized controlled trials to evaluate its impact on cardiovascular outcomes. Biomarkers predictive of treatment response to PD-1 inhibitors are needed to guide patient selection and optimize therapeutic outcomes. Future studies should focus on identifying reliable biomarkers, such as PD-1 expression levels, immune cell subsets, or circulating inflammatory markers, that can predict cardiovascular risk and response to PD-1-based immunotherapy in HIV-infected individuals.[141-150]

Combination regimens incorporating PD-1 blockade with standard cardiovascular medications or other immunotherapeutic agents may enhance treatment efficacy and overcome resistance mechanisms. Synergistic effects may be achieved by targeting multiple pathways involved in cardiovascular pathology, such as inflammation, lipid metabolism, and endothelial function. Clinical trials investigating combination therapies are warranted to evaluate their potential benefits in HIV-associated CVD. Further mechanistic studies are needed to elucidate the specific role of PD-1 signaling in HIV-associated CVD. Understanding the cellular and molecular mechanisms underlying PD-1-mediated vascular pathology, including its effects on endothelial cells, immune cells, and vascular smooth muscle cells, will inform the development of targeted therapies and biomarkers for cardiovascular risk stratification. Longitudinal studies with long-term follow-up are essential to assess the safety and durability of PD-1-based immunotherapy in HIV-infected individuals with cardiovascular complications. Monitoring for immune-related adverse events and cardiovascular events, including myocardial infarction, stroke, and heart failure, is crucial to ensure the optimal management of patients receiving PD-1 blockade.[151-170]

Conclusion

The programmed cell death protein 1 (PD-1) signaling pathway has emerged as a key player in the pathogenesis of HIV-associated cardiovascular disease (CVD), offering promising therapeutic implications and avenues for future research. Chronic inflammation, immune dysregulation, and endothelial dysfunction contribute to the development and progression of cardiovascular complications in individuals living with HIV/AIDS. PD-1 signaling influences vascular

inflammation, endothelial function, atherosclerosis, and myocardial injury, highlighting its multifaceted role in cardiovascular pathology.

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