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Neutrophil-Mediated Vascular Damage and Pulmonary Hypertension in HIV-Associated Cardiovascular Diseases: A Review

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Abstract

Pulmonary hypertension (PH) remains a significant complication in individuals living with human immunodeficiency virus (HIV), contributing to increased morbidity and mortality. Emerging evidence suggests a pivotal role of neutrophil-mediated vascular damage in the pathogenesis of PH in HIV-associated cardiovascular diseases. This review aims to consolidate current knowledge regarding the intricate interplay between neutrophils, vascular damage, and the development of pulmonary hypertension in HIV patients. We delve into the mechanisms by which neutrophils contribute to vascular injury, inflammation, and remodeling in the pulmonary vasculature, leading to PH. Furthermore, we explore the specific alterations in neutrophil phenotype and function observed in HIV-infected individuals and their potential implications for pulmonary vascular pathology. Additionally, this review discusses the clinical implications of neutrophil-mediated vascular damage in HIV-associated pulmonary hypertension, including diagnostic approaches, therapeutic strategies, and areas warranting further research. Overall, elucidating the role of neutrophils in vascular damage associated with pulmonary hypertension in HIV patients could offer novel insights into disease pathogenesis and unveil potential therapeutic targets for mitigating PH-related complications in this population.

Keywords: *Neutrophils, vascular damage, pulmonary hypertension, HIV, cardiovascular diseases*

Introduction

Pulmonary hypertension (PH) presents a formidable challenge in the realm of HIV-associated cardiovascular diseases, significantly contributing to heightened morbidity and mortality rates

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among affected individuals. While advancements in antiretroviral therapy have notably improved the life expectancy of HIV patients, the incidence of PH remains disproportionately elevated in this population compared to the general populace. Despite considerable efforts to elucidate the multifaceted etiology of PH in HIV, the precise mechanisms underpinning its pathogenesis remain incompletely understood. However, emerging evidence suggests a central role for neutrophil-mediated vascular damage in driving the progression of PH in HIV-infected individuals. Neutrophils, traditionally regarded as the frontline defenders against microbial invaders, have garnered increasing attention for their pivotal involvement in orchestrating inflammatory responses and tissue damage in various pathological conditions. In the context of HIV infection, dysregulation of neutrophil function and phenotype has been widely documented, with implications extending beyond conventional immune dysfunction. Notably, recent studies have implicated neutrophils as key players in the pathogenesis of vascular damage, particularly within the pulmonary vasculature, thereby shedding light on their potential contribution to the development and progression of PH in HIV patients.[1-25]

The intricate interplay between neutrophils and the pulmonary vasculature in HIV-associated PH underscores the need for a comprehensive understanding of neutrophil biology and function in this context. Neutrophils possess a diverse repertoire of effector mechanisms, including the release of reactive oxygen species, proteases, and cytokines, all of which have been implicated in vascular injury and endothelial dysfunction. Moreover, emerging evidence suggests that neutrophil-derived mediators may drive aberrant vascular remodeling processes, further exacerbating PH in HIV-infected individuals. Despite the growing recognition of neutrophils as key contributors to vascular damage and PH in HIV, several knowledge gaps persist, warranting further investigation. Clarifying the specific mechanisms by which neutrophils interact with the pulmonary vasculature and deciphering the factors governing their dysregulated behavior in HIV infection are critical for advancing our understanding of PH pathogenesis. Additionally, translating these insights into clinically relevant diagnostic and therapeutic strategies represents a pressing need in improving outcomes for HIV patients burdened by PH. This review seeks to address these gaps by synthesizing current evidence and highlighting potential avenues for future research and clinical intervention in the realm of neutrophil-mediated vascular damage and PH in HIV-associated cardiovascular diseases.[26-51]

Neutrophil Biology and Function

Neutrophils, the most abundant type of white blood cells in circulation, play a central role in the innate immune response against microbial pathogens. Originating from the bone marrow, neutrophils are rapidly recruited to sites of infection or tissue injury, where they execute various effector functions aimed at pathogen clearance and tissue repair. Central to their antimicrobial activity is phagocytosis, wherein neutrophils engulf and degrade invading microorganisms through the action of antimicrobial proteins and reactive oxygen species generated within specialized intracellular compartments known as phagosomes. Additionally, neutrophils release neutrophil extracellular traps (NETs), web-like structures composed of chromatin and antimicrobial proteins,

to ensnare and neutralize pathogens extracellularly. Beyond their role in host defense, neutrophils contribute to the regulation of inflammatory responses and tissue homeostasis through the release of cytokines, chemokines, and lipid mediators. These soluble factors facilitate communication with other immune cells and orchestrate the recruitment and activation of additional inflammatory cells to the site of infection or injury. Furthermore, neutrophils can modulate the adaptive immune response by interacting with dendritic cells, T cells, and B cells, thereby influencing the development and resolution of immune reactions.[52-56]

In the context of HIV infection, dysregulation of neutrophil biology and function has been extensively documented. HIV-induced alterations in cytokine profiles, cellular signaling pathways, and metabolic processes can profoundly impact neutrophil phenotype and behavior. Notably, HIV infection is associated with increased neutrophil activation and turnover, as evidenced by elevated levels of circulating neutrophil activation markers and heightened oxidative burst activity. These aberrant neutrophil responses contribute to chronic immune activation and tissue damage observed in HIV-infected individuals. Moreover, HIV-mediated dysregulation of neutrophil function extends beyond traditional immune parameters, encompassing effects on non-immune cells and tissues. For instance, HIV-infected neutrophils exhibit enhanced adhesion to endothelial cells and augmented release of pro-inflammatory cytokines, which can promote endothelial dysfunction and vascular inflammation. Additionally, neutrophil-derived factors have been implicated in the pathogenesis of HIV-associated comorbidities, including cardiovascular diseases and neurocognitive disorders, underscoring the multifaceted impact of neutrophils in HIV pathophysiology.[57-78]

Neutrophil-Mediated Vascular Damage

Neutrophils, traditionally recognized for their role in combating microbial infections, also play a significant role in mediating vascular damage in various pathological conditions, including those associated with human immunodeficiency virus (HIV) infection. Upon activation, neutrophils release an array of cytotoxic molecules and inflammatory mediators that can directly injure the endothelial lining of blood vessels and disrupt vascular integrity. One of the hallmark features of neutrophil-mediated vascular damage is the generation of reactive oxygen species (ROS) through the respiratory burst pathway. ROS, such as superoxide anion and hydrogen peroxide, can inflict oxidative stress on endothelial cells, leading to lipid peroxidation, DNA damage, and impaired endothelial function. In addition to ROS production, neutrophils release proteolytic enzymes, such as matrix metalloproteinases (MMPs) and elastase, which can degrade extracellular matrix components and compromise the structural integrity of blood vessel walls. MMP-mediated degradation of vascular basement membranes and interstitial collagen contributes to endothelial dysfunction, vascular leakage, and the formation of atherosclerotic plaques. Moreover, neutrophil-derived elastase can directly damage endothelial cells and promote thrombus formation by cleaving von Willebrand factor and tissue factor pathway inhibitor.[79-100]

Furthermore, neutrophils actively participate in the inflammatory response within the vascular microenvironment by releasing pro-inflammatory cytokines, chemokines, and lipid mediators. These soluble factors not only recruit additional immune cells to the site of vascular injury but also amplify local inflammation and perpetuate tissue damage. Neutrophil-derived cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β), stimulate endothelial cell activation and upregulate the expression of adhesion molecules, facilitating leukocyte adhesion and transendothelial migration. Additionally, neutrophil-derived lipid mediators, including leukotrienes and prostaglandins, promote vasodilation, vascular permeability, and leukocyte recruitment, further exacerbating vascular damage and inflammation. The consequences of neutrophil-mediated vascular damage extend beyond local tissue injury and inflammation, contributing to the pathogenesis of systemic vascular diseases, such as atherosclerosis, thrombosis, and pulmonary hypertension (PH). In the context of HIV infection, dysregulated neutrophil activation and vascular damage are implicated in the development of HIV-associated PH, a severe complication characterized by elevated pulmonary arterial pressure and progressive right heart failure. Neutrophil-induced endothelial dysfunction, vascular remodeling, and thromboinflammation collectively contribute to the pathophysiology of HIV-associated PH, highlighting the critical role of neutrophils in driving vascular complications in HIV-infected individuals.[101-109]

Neutrophil Infiltration and Inflammation in Pulmonary Vasculature

In the context of pulmonary hypertension (PH) associated with HIV, neutrophil infiltration and inflammation within the pulmonary vasculature emerge as critical pathological processes driving disease progression.[110-112] Neutrophil infiltration into the pulmonary vasculature is facilitated by various chemotactic factors, including cytokines, chemokines, and damage-associated molecular patterns (DAMPs), which are released in response to endothelial injury, infection, or inflammation. These factors orchestrate the recruitment of circulating neutrophils to the pulmonary vascular bed, where they contribute to vascular damage and remodeling through a multitude of effector mechanisms. Upon extravasation into the pulmonary tissue, neutrophils interact with endothelial cells, platelets, and other immune cells, triggering a cascade of inflammatory events. Neutrophils adhere to activated endothelial cells via adhesion molecules such as selectins and integrins, facilitating their migration across the endothelial barrier into the perivascular space. Once in the pulmonary interstitium and perivascular areas, neutrophils release a plethora of pro-inflammatory mediators, including cytokines (e.g., tumor necrosis factor-alpha, interleukin-6), chemokines (e.g., interleukin-8), and lipid mediators (e.g., leukotrienes), exacerbating local inflammation and tissue damage.

In addition to their role in promoting inflammation, neutrophils contribute to endothelial dysfunction, a hallmark feature of PH pathophysiology. Neutrophil-derived reactive oxygen species (ROS) and proteases can directly damage endothelial cells, impairing their barrier function and disrupting vascular homeostasis.1[12] Moreover, neutrophil-induced release of pro-inflammatory cytokines and vasoactive substances can further compromise endothelial integrity

and promote vasoconstriction, exacerbating pulmonary vascular remodeling and hypertension. Furthermore, neutrophils can engage in crosstalk with other immune cells and structural cells within the pulmonary vasculature, amplifying the inflammatory response and potentiating vascular damage. Neutrophil-platelet interactions, for instance, facilitate platelet activation and aggregation, leading to the release of vasoactive and pro-thrombotic factors that promote vascular dysfunction and thrombus formation. Additionally, neutrophils can stimulate resident immune cells, such as macrophages and dendritic cells, to produce additional inflammatory mediators, thereby perpetuating the inflammatory cascade and fostering a pro-inflammatory microenvironment conducive to vascular remodeling and PH progression.

Neutrophil-Driven Vascular Remodeling

In the intricate pathogenesis of pulmonary hypertension (PH) associated with HIV, neutrophils contribute significantly to vascular remodeling, a hallmark feature of the disease.[113] Vascular remodeling encompasses structural alterations in the pulmonary vasculature, including vascular wall thickening, smooth muscle cell proliferation, and fibrosis, ultimately culminating in increased pulmonary vascular resistance and right ventricular strain. Neutrophil-driven vascular remodeling in the context of HIV-associated PH involves complex interactions between neutrophils, resident vascular cells, and extracellular matrix components, orchestrating pathological changes within the pulmonary vasculature. Central to neutrophil-mediated vascular remodeling is the release of bioactive molecules that modulate the behavior of vascular cells, including endothelial cells, smooth muscle cells, and fibroblasts. Neutrophils secrete a myriad of cytokines, chemokines, growth factors, and matrix metalloproteinases (MMPs) that promote cellular proliferation, migration, and extracellular matrix remodeling. For instance, neutrophil-derived transforming growth factor-beta (TGF-β) stimulates fibroblast activation and collagen deposition, contributing to pulmonary vascular fibrosis and stiffening. Similarly, MMPs released by neutrophils facilitate matrix degradation and remodeling, promoting vascular remodeling and destabilizing the vascular architecture.

Moreover, neutrophils interact directly with vascular cells through cell-cell contact and paracrine signaling, further exacerbating vascular remodeling processes. Neutrophil-derived reactive oxygen species (ROS) and proteases induce endothelial dysfunction, promoting the expression of adhesion molecules and pro-inflammatory cytokines, which in turn enhance leukocyte recruitment and exacerbate vascular inflammation. Additionally, neutrophil-derived factors such as platelet-derived growth factor (PDGF) and vascular endothelial growth factor (VEGF) stimulate smooth muscle cell proliferation and migration, leading to medial hypertrophy and neointima formation.[114] Furthermore, neutrophils contribute to vascular remodeling indirectly through their interactions with platelets and other immune cells within the pulmonary vasculature. Neutrophil-platelet aggregates release vasoactive and mitogenic factors that promote endothelial dysfunction and smooth muscle cell proliferation, fostering vascular remodeling. Additionally, neutrophils can activate resident immune cells, such as macrophages and dendritic cells, to produce pro-inflammatory cytokines and chemokines that perpetuate vascular inflammation and remodeling.

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Clinical Implications and Therapeutic Targets

Understanding the intricate interplay between neutrophils and pulmonary vascular pathology in HIV-associated pulmonary hypertension (PH) holds significant clinical implications for the management of affected individuals.[115] By elucidating the role of neutrophils in driving vascular damage and remodeling, clinicians can potentially identify novel diagnostic markers and therapeutic targets to improve patient outcomes. Additionally, targeting neutrophil-mediated pathways may offer adjunctive therapeutic strategies to complement existing treatments for PH in HIV-infected individuals. One of the clinical implications of neutrophil involvement in HIV-associated PH lies in the realm of disease diagnosis and monitoring. Biomarkers associated with neutrophil activation and inflammation, such as neutrophil elastase, myeloperoxidase, and circulating neutrophil counts, may serve as valuable indicators of disease severity and progression. Incorporating these biomarkers into clinical algorithms for PH risk stratification and monitoring could enhance early detection and enable timely intervention in HIV-infected individuals at risk of developing PH.

Furthermore, targeting neutrophil-driven pathways implicated in pulmonary vascular pathology holds promise for the development of novel therapeutic interventions. Pharmacological agents that modulate neutrophil activation, migration, and effector functions, such as neutrophil elastase inhibitors, chemokine receptor antagonists, and ROS scavengers, represent potential therapeutic targets for mitigating inflammation-driven vascular damage and remodeling in HIV-associated PH. Additionally, strategies aimed at disrupting neutrophil-platelet interactions or inhibiting neutrophil-derived mediators involved in endothelial dysfunction and smooth muscle cell proliferation may attenuate PH progression in HIV-infected individuals.[116] In addition to pharmacological interventions, non-pharmacological approaches targeting systemic inflammation and immune dysregulation may also hold therapeutic potential in HIV-associated PH. Strategies aimed at reducing HIV-associated immune activation and viral replication, such as antiretroviral therapy and immunomodulatory agents, could indirectly mitigate neutrophil-mediated vascular damage and inflammation, thereby attenuating PH progression. Furthermore, lifestyle modifications, including smoking cessation, exercise training, and dietary interventions, may complement pharmacological therapies by targeting underlying risk factors and promoting vascular health in HIV-infected individuals with PH. Despite the promising therapeutic avenues outlined above, several challenges and knowledge gaps remain in translating neutrophil-targeted therapies into clinical practice. Further research is warranted to elucidate the precise mechanisms underlying neutrophil-mediated vascular damage and remodeling in HIV-associated PH, as well as to evaluate the safety and efficacy of novel therapeutic agents targeting neutrophil-driven pathways. Additionally, clinical trials assessing the impact of adjunctive neutrophil-targeted therapies on PH outcomes in HIV-infected individuals are needed to validate their utility in real-world settings and inform evidence-based treatment guidelines.

Conclusion

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The intricate interplay between neutrophils and the pulmonary vasculature emerges as a pivotal determinant in the pathogenesis of pulmonary hypertension (PH) associated with HIV. Neutrophils, traditionally regarded as frontline defenders against microbial invaders, exhibit multifaceted roles in promoting vascular damage, inflammation, and remodeling within the pulmonary circulation, ultimately contributing to the development and progression of PH in HIV-infected individuals. The infiltration and activation of neutrophils within the pulmonary vasculature contribute to endothelial dysfunction, inflammation, and vascular remodeling, culminating in increased pulmonary vascular resistance and right ventricular strain. Neutrophil-derived bioactive molecules, including cytokines, chemokines, reactive oxygen species, and proteases, modulate the behavior of vascular cells and extracellular matrix components, perpetuating a pro-inflammatory and pro-fibrotic microenvironment conducive to PH pathophysiology.

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