Elite Journal of HIV. Volume 2 issue 3(2024), Pp. 14-26
https://epjournals.com/journals/EJHIV

Assessing Platelet Functionality in HIV Patients Receiving Antiretroviral Therapy: Implications for Risk Assessment

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Abstract

Antiretroviral therapy (ART) has significantly improved the prognosis of HIV-infected individuals, yet concerns persist regarding its impact on platelet function and subsequent risks of thrombosis and bleeding. This review article explores the intricate relationship between ART and platelet functionality, aiming to provide insights into the mechanisms, clinical implications, and strategies for risk assessment in HIV patients. Chronic inflammation, immune dysregulation, and endothelial dysfunction associated with HIV infection contribute to platelet hyperreactivity and enhanced thrombotic potential. Moreover, certain antiretroviral drugs, notably protease inhibitors and nucleoside reverse transcriptase inhibitors, have been implicated in platelet activation and impaired aggregation, possibly through direct drug toxicity or alterations in platelet signaling pathways. Platelet dysfunction in HIV patients receiving ART has significant clinical implications, including an elevated risk of cardiovascular events, venous thromboembolism, and bleeding complications. Effective risk assessment strategies should encompass evaluation of individual patient characteristics, ART regimen, viral load, CD4 count, and traditional cardiovascular risk factors. Biomarkers of platelet activation and function, such as platelet aggregometry and soluble markers, offer potential utility in risk stratification and therapeutic decision-making. This review underscores the importance of understanding the complex interplay between HIV infection, ART, and platelet function, and highlights the need for tailored management approaches to mitigate adverse cardiovascular and thrombotic outcomes in HIV patients.

Keywords: *HIV, antiretroviral therapy, platelet function, platelet aggregation, thrombosis, bleeding, cardiovascular risk*

Citation: Obeagu EI, Obeagu GU. Assessing Platelet Functionality in HIV Patients Receiving Antiretroviral Therapy: Implications for Risk Assessment. Elite Journal of HIV, 2024; 2(3): 14-26

Introduction

Antiretroviral therapy (ART) has transformed the landscape of HIV/AIDS management, drastically reducing morbidity and mortality associated with the infection. However, as the life expectancy of HIV-infected individuals has increased, attention has turned towards addressing the long-term complications and comorbidities associated with both the virus and its treatment. Among these concerns, alterations in platelet function have emerged as a topic of interest, given their potential implications for cardiovascular health and thrombotic risk. Platelets, traditionally recognized for their role in hemostasis, are increasingly understood to be intricately involved in inflammation, immune response modulation, and vascular health. Consequently, any perturbations in platelet function may have far-reaching consequences for individuals living with HIV. The relationship between HIV infection, ART, and platelet function is complex and multifaceted. HIV infection itself is associated with chronic immune activation and inflammation, which can lead to platelet hyperreactivity and increased thrombotic propensity. Furthermore, the introduction of ART, while essential for viral suppression and immune reconstitution, has been implicated in various hematological abnormalities, including alterations in platelet count, morphology, and function. Both HIV infection and certain antiretroviral drugs may directly impact platelet biology through diverse mechanisms, such as direct viral effects, drug toxicity, metabolic disturbances, and dysregulation of platelet signaling pathways.[1-31]

The clinical significance of platelet dysfunction in HIV patients receiving ART cannot be overstated. Thrombotic events, such as myocardial infarction, stroke, and venous thromboembolism, represent leading causes of morbidity and mortality in this population. Conversely, bleeding complications, though less common, remain a concern, particularly in the context of thrombocytopenia and coagulation disorders associated with HIV and certain antiretroviral agents. Effective risk assessment and management strategies necessitate a comprehensive understanding of the factors contributing to platelet dysfunction in HIV-infected individuals. Moreover, the growing recognition of platelets as key mediators of inflammation and immune response underscores their potential role as biomarkers and therapeutic targets in HIV/AIDS. This review seeks to explore the current state of knowledge regarding platelet function in HIV patients receiving ART, with a focus on elucidating mechanisms, assessing clinical implications, and guiding risk stratification and management approaches. Through a deeper understanding of platelet biology in the context of HIV infection and ART, clinicians and researchers can work towards optimizing cardiovascular health and minimizing thrombotic risks in this vulnerable population.[32-50]

Mechanisms of Platelet Dysfunction in HIV and ART

HIV infection is characterized by chronic immune activation and systemic inflammation, which can directly impact platelet function. Elevated levels of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β), can activate platelets and promote their adhesion and aggregation. Persistent immune activation also leads to endothelial dysfunction, further exacerbating platelet activation and thrombotic potential. HIV has

been shown to directly interact with platelets and alter their function. The virus can bind to platelet surface receptors, such as CD4 and CCR5, leading to platelet activation and release of inflammatory mediators. Additionally, HIV proteins, such as Tat and gp120, may induce platelet activation and aggregation through various signaling pathways, contributing to thrombotic complications. Certain antiretroviral drugs, particularly protease inhibitors (PIs) and nucleoside reverse transcriptase inhibitors (NRTIs), have been associated with hematological abnormalities, including platelet dysfunction. PIs, such as ritonavir and lopinavir, can inhibit platelet aggregation and alter platelet membrane structure and function. NRTIs, such as zidovudine (AZT) and stavudine (d4T), may cause mitochondrial toxicity and impair platelet production and function. HIV infection and ART can lead to metabolic abnormalities, such as dyslipidemia, insulin resistance, and hyperglycemia, which may indirectly affect platelet function. Dyslipidemia, in particular, has been associated with increased platelet activation and aggregation, potentially contributing to cardiovascular risk in HIV-infected individuals. HIV and ART may dysregulate various platelet signaling pathways involved in hemostasis, thrombosis, and inflammation. Activation of protein kinase C (PKC) and mitogen-activated protein kinase (MAPK) pathways, as well as dysregulation of cyclic AMP (cAMP) and cyclic GMP (cGMP) signaling, have been implicated in platelet dysfunction associated with HIV and certain antiretroviral drugs.[51-78]

Clinical Implications and Risk Assessment

Platelet dysfunction in HIV-infected individuals receiving antiretroviral therapy (ART) has significant clinical implications, particularly in terms of thrombotic and bleeding risks. HIV-infected individuals have an increased risk of cardiovascular disease and venous thromboembolism compared to the general population. Platelet dysfunction, characterized by hyperreactivity and enhanced thrombotic potential, may further exacerbate these risks. Thrombotic events, such as myocardial infarction, stroke, and deep vein thrombosis, represent leading causes of morbidity and mortality in HIV patients receiving ART. Therefore, thorough risk assessment for thrombotic events is essential, taking into account traditional cardiovascular risk factors, HIV-specific factors (e.g., viral load, CD4 count), and antiretroviral drug effects on platelet function. While thrombotic events are a concern, bleeding complications also warrant attention, particularly in the context of thrombocytopenia and coagulation disorders associated with HIV and certain antiretroviral agents. Platelet dysfunction, manifested by impaired aggregation and clot formation, may predispose HIV-infected individuals to bleeding episodes, ranging from minor mucocutaneous bleeding to severe gastrointestinal or intracranial hemorrhage. Therefore, risk assessment for bleeding complications should be integrated into clinical decision-making, considering factors such as platelet count, coagulation profile, and concurrent medications (e.g., nonsteroidal anti-inflammatory drugs, anticoagulants).[79-99]

Effective risk assessment for thrombotic and bleeding complications in HIV patients receiving ART requires a multidimensional approach. This involves evaluating individual patient characteristics, including demographics, comorbidities, HIV disease status, and antiretroviral drug regimen. Laboratory assessments, such as platelet function tests (e.g., aggregometry, thromboelastography) and biomarkers of platelet activation (e.g., soluble P-selectin, thromboxane

B2), may provide valuable insights into platelet function and help stratify thrombotic and bleeding risks. Based on risk assessment findings, management strategies can be tailored to mitigate thrombotic and bleeding risks in HIV-infected individuals receiving ART. This may include optimizing antiretroviral drug selection, monitoring for hematological abnormalities, and implementing preventive measures, such as antiplatelet therapy or anticoagulation, as appropriate. Close collaboration between infectious disease specialists, hematologists, cardiologists, and other healthcare providers is essential for comprehensive risk assessment and individualized management of HIV patients at risk for thrombotic and bleeding complications.[100-113]

Conclusion

Platelet dysfunction represents a significant clinical concern in HIV-infected individuals receiving antiretroviral therapy (ART), with implications for both thrombotic and bleeding risks. The complex interplay between HIV infection, ART, and platelet function underscores the importance of comprehensive risk assessment strategies to optimize patient management and reduce adverse outcomes. Thorough evaluation of individual patient characteristics, including demographics, HIV disease status, antiretroviral drug regimen, and laboratory parameters, is essential for stratifying thrombotic and bleeding risks accurately. While HIV-infected individuals are at increased risk of cardiovascular disease and venous thromboembolism, platelet dysfunction may further exacerbate these risks. Conversely, bleeding complications, particularly in the context of thrombocytopenia and coagulation disorders, remain a concern. Tailored management strategies, guided by risk assessment findings, may include optimizing antiretroviral drug selection, monitoring for hematological abnormalities, and implementing preventive measures such as antiplatelet therapy or anticoagulation when indicated.

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Citation: Obeagu EI, Obeagu GU. Assessing Platelet Functionality in HIV Patients Receiving Antiretroviral Therapy: Implications for Risk Assessment. Elite Journal of HIV, 2024; 2(3): 14-26

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