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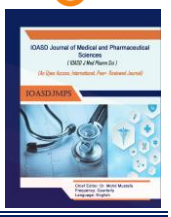

Platelets and People: Comparing Thrombocyte Patterns in African and Russian HIV Cohorts– A Review

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ABSTRACT

Platelet abnormalities are common hematological manifestations of HIV infection, yet their patterns vary across regions due to differences in comorbidities, treatment access, and population-specific physiological baselines. Africa and Russia represent two distinct HIV epidemics with unique epidemiological drivers and hematologic reference norms. Understanding how platelet indices differ between these settings is essential for improving diagnostic accuracy and patient management. This review synthesizes evidence on thrombocyte patterns among HIV-positive cohorts in Africa and Russia, with emphasis on thrombocytopenia, regional baseline platelet reference ranges, and the influence of co-infections and treatment status. A narrative review was conducted through searches in PubMed, Scopus, Web of Science, and AJOL for studies published between 1990 and 2025. Observational and cohort studies reporting platelet indices in HIV-infected adults or children from African or Russian populations were included. Findings show that thrombocytopenia is more prevalent in African HIV cohorts, often driven by immune-mediated platelet destruction, opportunistic infections, and endemic co-infections such as malaria and tuberculosis. Russian cohorts exhibit platelet abnormalities more frequently linked to late HIV diagnosis, hepatitis C co-infection, and substance use–related complications. Regional platelet reference ranges differ considerably, with African populations generally showing lower baseline values than Russian or global standards, impacting diagnostic thresholds and disease classification. Platelet patterns in HIV infection vary markedly between Africa and Russia due to distinct epidemiologic and healthcare factors. Context-specific reference ranges and diagnostic criteria are essential for accurate monitoring and improved clinical decision-making.

Keywords: HIV, thrombocytopenia, platelets, Africa, Russia.

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INTRODUCTION

Platelet abnormalities are among the most frequent hematological disturbances observed in individuals living with HIV, reflecting the complex interplay between viral replication, immune dysregulation, co-infections, and antiretroviral therapy (ART) [1-3]. Thrombocytopenia, commonly defined as a platelet count of <150,000/μL, occurs across all stages of infection and may serve as an early clinical indicator of disease progression or systemic inflammatory activity [4-6]. However, the expression, severity, and clinical implications of HIV-associated thrombocytopenia differ substantially by region, influenced by population-specific hematologic baselines, comorbidity profiles, and healthcare access. Africa and Russia represent two contrasting HIV landscapes where these differences are particularly pronounced [7-10]. Sub-Saharan Africa continues to bear the highest global HIV burden, shaped by diverse epidemiological drivers such as high rates of tuberculosis, malaria, chronic viral infections, and nutritional deficiencies [11-13]. These conditions are known to alter bone marrow function and immune pathways, thereby intensifying the risk of thrombocytopenia. Furthermore, chronic inflammation, delayed diagnosis, and variable access to ART contribute to a wide spectrum of thrombocyte abnormalities. Several African hematologic reference studies report lower platelet baseline ranges compared to global norms, emphasizing the need for region-specific diagnostic thresholds [14-16].

In contrast, Russia's HIV epidemic is heavily influenced by late presentation, injection drug use, hepatitis C co-infection, and rapid urban transmission patterns [17-18]. These factors introduce unique hematologic challenges, including mixed-etiology thrombocytopenia arising from hepatic dysfunction, alcohol-related marrow suppression, and chronic systemic inflammation. Russian populations generally exhibit higher baseline platelet ranges compared to many African cohorts, creating a distinct reference context for interpreting thrombocyte abnormalities [19-20]. Despite increasing recognition of regional variation in HIV-associated hematologic parameters, comparative analyses between Africa and Russia remain limited [21-22]. Most existing evidence focuses on single-country cohorts, and few studies integrate thrombocyte patterns with the broader socio-clinical and epidemiologic determinants unique to each region. A clearer comparative understanding is essential for refining diagnostic strategies, contextualizing platelet thresholds, and improving the interpretation of thrombocytopenia as a biomarker of HIV disease activity [23-24]. This review therefore synthesizes current evidence on thrombocyte patterns among HIV-positive populations from Africa and Russia, highlighting regional platelet reference values, major clinical determinants, and the role of ART and co-infections. By contextualizing thrombocytopenia within these differing health systems and epidemiologic environments, this work aims to inform tailored diagnostic approaches and support more precise clinical decision-making across diverse HIV settings.

METHODS

This narrative review employed an integrative synthesis approach to compare platelet abnormalities—particularly thrombocytopenia—among HIV-infected populations in Africa and Russia. The review methodology was designed to capture a broad yet rigorous overview of hematologic patterns across diverse settings with differing epidemiological profiles, healthcare capacities, and HIV treatment landscapes. A comprehensive literature search was conducted across major scientific databases, including PubMed, Scopus, Web of Science, and African Journals Online (AJOL), covering publications from January 1990 to October 2025 (Figure 1). These dates were selected to encompass both the pre-antiretroviral therapy (pre-ART) era and the modern ART period, thereby allowing for an assessment of temporal changes in platelet indices. Search terms combined keywords and MeSH headings such as *"HIV," "thrombocytopenia," "platelet count," "thrombocyte," "hematological abnormalities," "Africa,"* and *"Russia."* Boolean operators (AND/OR) were used to refine results.

Inclusion criteria prioritized observational studies, cohort analyses, cross-sectional investigations, and regional surveillance reports that provided quantitative or qualitative descriptions of platelet parameters among HIV-positive adults or children in African or Russian populations. Studies focusing exclusively on opportunistic infections or antiviral therapy outcomes were included only if platelet metrics were explicitly reported. Exclusion criteria eliminated reviews, commentaries, case reports, and studies with insufficient methodological clarity or incomplete hematologic data. All eligible articles were screened manually to extract key variables related to platelet indices, prevalence of thrombocytopenia, clinical determinants, ART exposure, disease stage, and regional contextual factors such as co-infection burdens or socioeconomic determinants of health. Where available, regional baseline platelet reference ranges for Africa and Russia were reviewed to contextualize the thresholds used for diagnosing thrombocytopenia.

The extracted data were synthesized narratively rather than meta-analyzed due to substantial heterogeneity in study designs, population characteristics, laboratory diagnostic thresholds, and clinical settings. Special emphasis was placed on identifying patterns that differentiate African HIV cohorts—often characterized by high burdens of co-infections such as malaria, tuberculosis, and nutritional deficiencies—from Russian cohorts, where HIV transmission dynamics, substance use patterns, and comorbidity profiles differ markedly. This methodological framework enabled a balanced, comparative analysis that integrates epidemiological evidence, clinical trends, and regional hematological baselines to elucidate how platelet patterns evolve across distinct geographic and sociodemographic contexts.

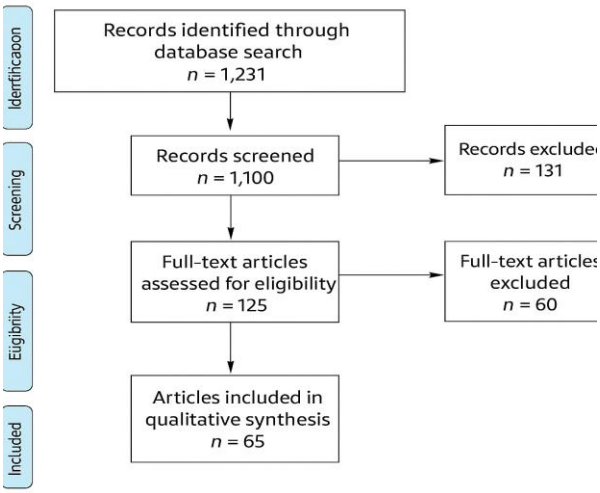

Figure 1: A PRISMA-style flow diagram illustrating the selection process

Limitations

This review has several important limitations that should be acknowledged when interpreting its findings. First, there is substantial heterogeneity in the available studies from both African and Russian settings, including differences in study designs, cohort characteristics, ART exposure, laboratory methods, and definitions of thrombocytopenia. These variations limit

the comparability of results across regions and may contribute to inconsistency in reported platelet patterns. Second, regional baseline platelet reference values are not uniformly established, particularly in many African countries, where population-based hematologic reference ranges remain scarce. As a result, applying the universal $<150 \times 10^9$/L cutoff may not fully reflect local physiological norms or epidemiologic realities. Third, several included studies were based on hospital-based or urban clinic populations, which may overrepresent individuals with advanced disease or comorbidities. This introduces a potential selection bias and limits the generalizability of findings to community-level populations. Fourth, co-infections such as malaria, tuberculosis, hepatitis C, and helminthic infections—conditions known to influence platelet counts—were inconsistently reported across studies. The lack of standardized adjustment for these comorbidities makes it difficult to isolate thrombocytopenia attributable solely to HIV.

Moreover, differences in diagnostic capacity and laboratory quality assurance systems between regions may have influenced platelet count measurements, introducing methodological bias that could not be fully accounted for. The review was also limited by the unequal volume and quality of published data from the two regions; Russian cohorts had more detailed information on ART regimens and liver-related comorbidities, while African studies frequently lacked comprehensive hematologic and clinical profiling. Finally, as a narrative rather than systematic review, this work may be subject to publication and selection bias, and despite efforts to capture a broad evidence base, some relevant studies may not have been identified. These limitations highlight the need for more harmonized, large-scale, and multi-regional studies that adopt standardized platelet measurement protocols and systematically account for comorbidities.

Thrombocytopenia in HIV: Pathophysiological Overview

Thrombocytopenia is a common hematological complication in people living with HIV and reflects a multifactorial pathophysiology involving impaired platelet production, immune-mediated destruction, increased peripheral consumption, and treatment- and comorbidity-related effects. Rather than representing a single disease entity, HIV-associated thrombocytopenia emerges from the dynamic interplay between viral replication, chronic immune activation, bone marrow dysfunction, endothelial perturbation, and co-existing infections or organ disease [25-26]. At the level of platelet production, HIV disrupts megakaryopoiesis through both direct and indirect mechanisms. Viral proteins and the inflammatory milieu characteristic of uncontrolled HIV infection impair megakaryocyte maturation and reduce thrombopoietin responsiveness. Chronic elevation of pro-inflammatory cytokines skews bone marrow homeostasis, suppressing effective platelet generation and contributing to broader cytopenias in advanced disease. These effects are accentuated in individuals with high viral loads, advanced immunosuppression, and nutritional deficiencies, underscoring the central role of systemic inflammation and marrow stress in platelet depletion [27-28].

Peripheral platelet destruction is driven in part by immune-mediated mechanisms that resemble secondary immune thrombocytopenia. Autoantibodies and immune complexes targeting platelet surface glycoproteins promote splenic clearance, while Fc receptor–mediated phagocytosis by activated macrophages further accelerates platelet loss. This immune component is closely linked to uncontrolled viral replication and heightened B-cell dysregulation, and it often improves with effective antiretroviral therapy, highlighting the reversibility of immune-mediated platelet destruction under virologic suppression [29]. Beyond quantitative depletion, HIV is associated with qualitative platelet dysfunction and heightened platelet activation. Endothelial activation, driven by microbial translocation and systemic inflammation, promotes platelet adhesion and aggregation within the microvasculature. Platelet–leukocyte interactions amplify inflammatory signaling and coagulation pathways, creating a paradoxical phenotype in which thrombocytopenia coexists with a prothrombotic, thromboinflammatory state. This duality has important clinical implications, as patients may face concurrent risks of bleeding and vascular complications [30].

Co-infections and comorbid conditions further shape thrombocytopenia in HIV through context-specific mechanisms. In settings with a high burden of tuberculosis, malaria, or chronic viral hepatitis, inflammatory platelet consumption, splenic sequestration, and bone marrow suppression act synergistically with HIV-related immune dysregulation. Liver disease, whether related to viral hepatitis or alcohol use, contributes through reduced thrombopoietin production, portal hypertension–associated hypersplenism, and impaired platelet survival. Nutritional deficiencies common in advanced HIV further compromise hematopoietic reserve, exacerbating thrombocytopenia and delaying recovery [31-32]. Antiretroviral therapy represents the most effective disease-modifying intervention for HIV-associated thrombocytopenia. Viral suppression leads to reductions in immune activation, restoration of marrow function, and attenuation of immune-mediated platelet destruction, resulting in platelet count recovery in the majority of patients. However, the hematologic benefit of ART may be attenuated by ongoing co-infections, liver disease, or the myelosuppressive effects of specific agents. Thus, platelet recovery is contingent not only on virologic control but also on integrated management of comorbidities and careful regimen selection [33].

Platelet Patterns in African HIV Cohorts

In sub-Saharan Africa, thrombocytopenia remains one of the most frequently encountered hematologic abnormalities among people living with HIV, reflecting the convergence of advanced disease presentation, high infectious burden, nutritional vulnerability, and variable access to sustained antiretroviral therapy. Platelet abnormalities in this setting are rarely attributable to HIV alone; rather, they emerge from layered biological and structural determinants that shape both baseline platelet distributions and disease-associated deviations [34]. A defining feature of African HIV cohorts is the high prevalence of co-infections that directly perturb platelet homeostasis. Tuberculosis and malaria, in particular, exert strong and often bidirectional effects on platelet dynamics through inflammatory consumption, splenic sequestration, and transient marrow suppression. Acute malaria is commonly associated with abrupt platelet declines, while chronic or disseminated tuberculosis promotes sustained inflammatory thrombopoiesis and peripheral platelet utilization. These infections frequently co-occur with advanced HIV, amplifying immune activation and accelerating platelet depletion. Chronic viral hepatitis and recurrent bacterial infections further contribute to platelet variability, especially in settings with limited diagnostic differentiation between overlapping etiologies of cytopenia [35].

Nutritional status is an important, and often underappreciated, modifier of platelet trends in African cohorts. Protein-energy malnutrition and deficiencies in folate, iron, and vitamin B12 compromise hematopoietic capacity, reducing marrow reserve and blunting platelet recovery during intercurrent illness or following antiretroviral initiation. These deficiencies interact with chronic inflammation to create a state of fragile thrombopoiesis, in which modest infectious or pharmacologic stressors precipitate clinically meaningful thrombocytopenia. Addressing micronutrient deficiencies has been associated with broader hematologic improvement, although evidence for platelet-specific benefit remains limited and context dependent [36]. Antiretroviral therapy has substantially reduced the prevalence and severity of HIV-associated thrombocytopenia across African settings, yet patterns of platelet recovery remain heterogeneous. Late presentation to care, intermittent treatment interruption, and drug–drug interactions with tuberculosis therapy continue to delay virologic suppression in many patients, prolonging immune-mediated platelet destruction and marrow dysfunction. Myelotoxic effects of older antiretroviral regimens and concomitant antimicrobial therapies may further complicate platelet trajectories, underscoring the importance of regimen selection and close hematologic monitoring during treatment transitions [37].

Health system constraints shape the clinical recognition and management of platelet abnormalities in African cohorts. Limited access to routine laboratory monitoring restricts early detection of declining platelet counts, while constrained diagnostic capacity complicates differentiation between HIV-related thrombocytopenia and infection- or nutrition-mediated cytopenias. As a result, thrombocytopenia is often identified late, when bleeding risk or multisystem illness has already emerged. Strengthening laboratory infrastructure and integrating platelet monitoring into routine HIV care pathways are therefore central to improving clinical outcomes [38]. Platelet patterns in African HIV cohorts reflect a context of high inflammatory burden, overlapping infectious exposures, and variable treatment continuity. Thrombocytopenia in this setting is best understood as a syndromic marker of systemic vulnerability rather than a discrete complication of HIV infection alone. Incorporating co-infection screening, nutritional assessment, and early, sustained antiretroviral therapy into care models is essential for stabilizing platelet trajectories and reducing downstream bleeding and thromboinflammatory risks [39-40].

Platelet Patterns in Russian HIV Cohorts

In Russian HIV cohorts, thrombocytopenia is frequently observed and is shaped by a distinct constellation of epidemiological and clinical modifiers, including high rates of hepatitis C virus (HCV) co-infection, alcohol use disorder, chronic liver disease, and tuberculosis. Unlike settings where advanced untreated HIV predominates as the primary driver of platelet decline, platelet abnormalities in this context often reflect mixed-etiology mechanisms in which HIV-related immune dysregulation intersects with hepatosplenic dysfunction and substance-related marrow toxicity [41]. HCV co-infection is a central determinant of platelet patterns in Russian cohorts. Chronic viral hepatitis contributes to thrombocytopenia through reduced hepatic thrombopoietin production, portal hypertension–associated hypersplenism, and immune-mediated platelet clearance. Progressive liver fibrosis further impairs platelet survival and amplifies peripheral sequestration, producing a pattern of chronic, moderate thrombocytopenia that may persist despite effective HIV viral suppression. Alcohol-related liver injury compounds these effects by exacerbating hepatic dysfunction and directly suppressing bone marrow activity, thereby reducing platelet production and impairing recovery following intercurrent illness [42-43].

Substance use and associated social vulnerabilities influence both the biology and clinical detection of thrombocytopenia. Injection drug use is linked to recurrent bacterial infections, episodic viremia, and delayed engagement in sustained antiretroviral therapy, all of which perpetuate immune activation and platelet consumption. In this setting, platelet abnormalities often coexist with broader cytopenias and inflammatory markers, reflecting cumulative marrow stress rather than isolated platelet pathology.

Tuberculosis, though less prevalent than in many African settings, remains an important co-morbidity that can precipitate inflammatory thrombocytopenia and complicate etiologic attribution [44-45]. Antiretroviral therapy has improved platelet outcomes in Russian cohorts, particularly among individuals achieving durable virologic suppression. However, the hematologic benefits of ART may be attenuated by persistent liver disease, ongoing alcohol exposure, and drug–drug interactions with hepatotoxic agents. As a result, platelet recovery is often incomplete, and chronic thrombocytopenia may persist even in virologically controlled patients. This underscores the importance of integrated HIV–hepatology care, careful ART regimen selection, and concurrent management of substance use disorders to optimize hematologic trajectories [46]. From a clinical and health system perspective, platelet monitoring in Russian HIV cohorts is generally supported by broader laboratory availability, facilitating etiologic workup for liver disease, hypersplenism, and co-infections. Nevertheless, marginalized populations face barriers to continuous care, leading to delayed detection and fragmented management of thrombocytopenia. Overall, platelet patterns in Russian cohorts reflect the cumulative impact of HIV, chronic viral hepatitis, liver disease, and substance-related comorbidities, highlighting the need for multidisciplinary care models that address both virologic control and the upstream determinants of thrombocyte dysregulation [47].

Comparative Insights and Ethnoracial Influences

Comparative analysis of African and Russian HIV cohorts highlights how thrombocyte patterns emerge from the interaction between shared HIV-related mechanisms and region-specific biological, epidemiological, and socio-structural contexts. While immune activation, impaired megakaryopoiesis, and immune-mediated platelet destruction represent common pathways across settings, the relative contribution of these mechanisms is differentially weighted by co-morbid disease burden, treatment continuity, and population-level health determinants [48]. In African cohorts, platelet trends are shaped predominantly by a high background of infectious and nutritional stressors superimposed on HIV-related immune dysregulation. Endemic co-infections such as tuberculosis and malaria amplify inflammatory platelet consumption and splenic sequestration, while recurrent bacterial infections perpetuate episodic platelet declines. Nutritional deficiencies and chronic anemia further constrain hematopoietic reserve, lowering the threshold at which systemic inflammation translates into clinically meaningful thrombocytopenia. These factors contribute to greater variability in platelet counts over time and a higher prevalence of thrombocytopenia among individuals with advanced or untreated HIV [49-50].

In contrast, platelet patterns in Russian cohorts are more strongly influenced by chronic viral hepatitis, alcohol-related liver disease, and substance use–associated comorbidities. Hepatosplenic dysfunction and reduced thrombopoietin production create a structural predisposition to sustained, moderate thrombocytopenia that may persist despite virologic control. This pattern reflects a shift from predominantly infection-driven platelet consumption toward organ disease–mediated impairment of platelet production and survival. Consequently, thrombocytopenia in this context often mirrors the trajectory of liver disease progression rather than HIV disease stage alone [51-52]. Ethnoracial and genetic diversity may further modulate platelet biology and inflammatory responses, although direct evidence linking specific genetic backgrounds to thrombocytopenia risk in HIV remains limited. In African populations, diverse genetic architectures and the coexistence of inherited hematologic traits may influence baseline hematologic parameters and inflammatory set points, potentially modifying platelet responses to infection and treatment. However, data directly connecting these factors to clinically significant thrombocytopenia are sparse, underscoring an important research gap [53-54].

From a diagnostic and interpretive perspective, these comparative patterns caution against uniform application of platelet thresholds and etiologic assumptions across regions. Similar platelet counts may reflect fundamentally different underlying pathophysiology—acute inflammatory consumption in high-infection-burden settings versus chronic organ disease–mediated suppression in settings dominated by viral hepatitis and alcohol-related liver injury. Recognizing these contextual differences is essential for accurate attribution of thrombocytopenia, appropriate prioritization of diagnostic workup, and tailoring of therapeutic strategies [55-56]. Comparative insights reinforce that thrombocytopenia in HIV is not a homogeneous entity but a context-dependent phenotype shaped by intersecting biological and social determinants. Integrating regional epidemiology, co-morbidity profiles, and population-specific baselines into clinical interpretation can improve diagnostic precision and support more equitable, context-sensitive HIV care (Table 1) [57].

Table 1: Comparative Overview of Thrombocyte Patterns in African and Russian HIV Cohorts

Feature	African HIV Cohorts	Russian HIV Cohorts
Baseline platelet counts	Generally lower; regional reference ranges often <150 × 10^9/L; influenced by endemic infections and nutritional status	Generally higher; closer to global standards; influenced by age and comorbidities

Feature	African HIV Cohorts	Russian HIV Cohorts
Prevalence of thrombocytopenia	Higher prevalence, particularly in ART-naïve or late-presenting individuals	Lower overall prevalence; thrombocytopenia often associated with liver disease or substance use
Primary contributing factors	Immune-mediated platelet destruction, opportunistic infections (TB, malaria), micronutrient deficiencies, chronic inflammation	Hepatitis C co-infection, alcohol use, hepatic dysfunction, late ART initiation
ART-related influences	Zidovudine-based regimens linked to bone marrow suppression; limited access in some regions affects platelet recovery	ART exposure variable; integrase inhibitor-based regimens increasingly used; liver toxicity contributes to thrombocyte alterations
Co-infections impacting platelets	Malaria, TB, helminthic infections, chronic viral infections (HBV, HCV in some regions)	HCV co-infection predominant; TB less common; alcohol-related liver disease contributes indirectly
Clinical manifestations	Increased risk of bleeding, mucocutaneous bruising; often coincides with advanced HIV or co-infections	Mild-to-moderate thrombocytopenia; often subclinical unless liver disease or advanced immunosuppression present
Laboratory considerations	Limited standardization in some regions; baseline population studies often scarce	Laboratory methods generally standardized in urban centers; regional variation exists in rural areas
Implications for diagnosis	Universal thrombocytopenia cutoff (<150 × 10^9/L) may overestimate burden; local reference ranges recommended	Baseline higher; thrombocytopenia often reflects comorbidity rather than HIV alone; context-specific interpretation required

Regional baselines and thresholds for thrombocytopenia: Africa and Russia

Thrombocytopenia is conventionally defined as a platelet count below **150 × 10^9/L (150,000/µL)** and this threshold is widely used in clinical practice and research. However, interpreting platelet counts in people living with HIV requires careful consideration of regional baseline values and the local epidemiologic context. Platelet distributions in a population are not invariant: they reflect genetic background, endemic infections, nutritional status, environmental exposures, laboratory methods, and local healthcare-seeking patterns. Consequently, a single universal cutoff can mask meaningful regional differences in both prevalence estimates and clinical interpretation. Below we synthesize factors that shape regional baselines and outline important considerations specific to African and Russian HIV cohorts [51-53].

Biological and environmental determinants of regional platelet baselines

Population-level platelet counts vary for biological and environmental reasons. Genetic factors (including ancestry-related hematologic norms), altitude, and sex/age distributions all influence normative platelet ranges. Endemic infectious diseases such as malaria, schistosomiasis, hepatitis viruses, and chronic parasitic infections can lower mean platelet counts through peripheral consumption, splenic sequestration, or bone marrow suppression. Nutritional deficiencies (iron, folate, vitamin B12) and chronic inflammatory states also modify platelet production and turnover. Finally, methodological differences — anticoagulant type, timing of sampling, and hematology analyzer calibration — produce inter-laboratory variability that can be misinterpreted as biological difference if not standardized [54-56].

Africa: contextual factors likely to lower platelet baselines and increase thrombocytopenia prevalence

In many parts of sub-Saharan Africa and other regions of the continent, several converging factors influence baseline platelet counts:

- **High burden of co-infections.** Malaria, chronic hepatitis, and tuberculosis are common and can directly or indirectly reduce platelet counts through consumption, hypersplenism, or marrow suppression.
- **Nutritional factors.** Micronutrient deficiencies and food insecurity are prevalent in some settings and may blunt thrombopoiesis.
- **Chronic inflammation.** Persistent immune activation in untreated or late-presenting HIV contributes to platelet activation and accelerated turnover.
- **Healthcare access and late presentation.** Delayed diagnosis and treatment of HIV and opportunistic infections amplify hematologic abnormalities at presentation.
- **Laboratory standardization.** Resource-limited settings may have less uniform laboratory calibration and fewer population-based hematologic reference studies, complicating cross-site comparisons [57].

Diagnostic Considerations

The diagnostic evaluation of thrombocytopenia in people living with HIV requires careful contextualization of platelet counts within regional

epidemiology, co-morbidity profiles, and treatment status. Because thrombocytopenia in HIV is multifactorial, accurate attribution depends not only on confirming low platelet counts but also on distinguishing HIV-related mechanisms from infection-, nutrition-, and organ disease–mediated causes that vary substantially across settings [51]. In sub-Saharan African cohorts, a central diagnostic challenge is differentiating HIV-associated thrombocytopenia from cytopenias driven by endemic co-infections. Malaria and tuberculosis frequently present with thrombocytopenia through inflammatory consumption and splenic sequestration, and these conditions may coexist with advanced HIV at presentation. Limited access to advanced laboratory diagnostics can complicate etiologic attribution, particularly where peripheral smear review, bone marrow assessment, and virologic monitoring are not routinely available. In this context, pragmatic diagnostic algorithms that integrate clinical syndromes (e.g., febrile illness, weight loss), basic laboratory indices (full blood count trends, markers of inflammation), and targeted screening for common co-infections are essential. Assessment of nutritional status and micronutrient deficiencies can further clarify contributory marrow suppression in patients with persistent or unexplained thrombocytopenia [52-53].

In Russian cohorts, diagnostic evaluation is more often centered on disentangling HIV-related platelet abnormalities from those driven by chronic liver disease and viral hepatitis. Routine liver function testing, viral hepatitis screening, and abdominal imaging to assess hepatosplenomegaly are integral to determining whether thrombocytopenia reflects hypersplenism, reduced thrombopoietin production, or ongoing immune-mediated platelet destruction. Tuberculosis screening remains relevant, particularly in individuals with constitutional symptoms or treatment failure. Given the high prevalence of alcohol use disorder in some populations, clinical assessment of substance use and its hematologic consequences is also critical to avoid misclassification of thrombocytopenia as primarily HIV-mediated [53-54]. Across both settings, interpretation of platelet counts should account for regional baseline reference ranges and longitudinal trends rather than isolated values. Modest reductions in platelet counts may have different clinical significance depending on background hematologic distributions and the presence of concurrent anemia or leukopenia. Serial monitoring allows differentiation between transient infection-related declines and persistent thrombocytopenia suggestive of immune-mediated or organ disease–related mechanisms. Where available, evaluation for secondary immune thrombocytopenia should be considered in patients with severe or refractory thrombocytopenia, particularly when platelet counts fail to recover with virologic suppression [55-56]. Diagnostic strategies for thrombocytopenia in HIV should be context-sensitive, prioritizing exclusion of prevalent co-infections, assessment of nutritional and hepatic contributors, and integration of treatment history and virologic status. Embedding platelet assessment within routine HIV care pathways and strengthening access to basic diagnostic tools can improve early identification, etiologic classification, and appropriate management of thrombocytopenia across diverse epidemiological settings [57].

Therapeutic Strategies and Regional Considerations

Effective management of thrombocytopenia in people living with HIV is anchored in sustained virologic suppression through ART, which addresses the central drivers of immune activation, marrow dysfunction, and immune-mediated platelet destruction. In most patients, platelet counts improve following viral suppression; however, the magnitude and durability of recovery depend on the timely initiation of ART, regimen tolerability, and the burden of co-morbid conditions. Therapeutic strategies must therefore extend beyond HIV control to encompass integrated management of infections, nutritional deficits, and organ disease that modulate platelet homeostasis [58-59]. In sub-Saharan African settings, regional considerations emphasize early diagnosis and uninterrupted ART in the context of high burdens of tuberculosis, malaria, and recurrent bacterial infections. Prompt identification and treatment of these co-infections reduce inflammatory platelet consumption and splenic sequestration, facilitating platelet recovery. ART regimen selection should account for baseline cytopenias and potential hematologic toxicity, particularly in patients receiving concomitant tuberculosis therapy. Nutritional assessment and targeted micronutrient supplementation may support hematopoietic resilience in malnourished populations, while strengthening laboratory capacity for routine platelet monitoring enables earlier detection of treatment-related cytopenias and intercurrent infection–associated declines. Community-based adherence support and decentralization of care further enhance treatment continuity, indirectly stabilizing platelet trajectories [60-61].

In Russian cohorts, therapeutic priorities are shaped by the high prevalence of chronic viral hepatitis, alcohol-related liver disease, and substance use disorders. Integrated HIV–hepatology care is essential to address hypersplenism, impaired thrombopoietin production, and progressive fibrosis that sustain thrombocytopenia despite virologic control. Selection of ART regimens with favorable hepatic safety profiles, avoidance of cumulative hepatotoxicity, and routine liver function monitoring are central to optimizing hematologic outcomes. Concurrent management of hepatitis C virus infection and structured interventions for alcohol use disorder can yield downstream benefits for platelet recovery by mitigating hepatic and inflammatory contributors to thrombocyte dysregulation [62-63]. Across regions, management of severe or symptomatic immune-mediated thrombocytopenia requires individualized escalation of care. Short-term

immunomodulatory therapies may be considered in patients with clinically significant bleeding or profound thrombocytopenia, while platelet transfusion remains reserved for life-threatening hemorrhage or procedural prophylaxis. These interventions should complement, not replace, definitive treatment of HIV and its co-morbid drivers. Embedding platelet surveillance within routine HIV care and adopting multidisciplinary, regionally tailored care models offer the most effective strategy for reducing bleeding risk, mitigating thromboinflammatory complications, and improving hematologic outcomes across diverse HIV populations [64].

CONCLUSION

Thrombocytopenia in HIV reflects a convergence of shared pathogenic mechanisms—immune activation, impaired megakaryopoiesis, immune-mediated platelet destruction, and thromboinflammatory consumption—modulated by region-specific epidemiology and health system contexts. Comparative analysis of African and Russian cohorts demonstrates that similar platelet phenotypes may arise from fundamentally different upstream drivers, ranging from infection- and nutrition-related inflammatory stress in high-burden African settings to chronic viral hepatitis, alcohol-related liver disease, and hepatosplenic dysfunction in Russian populations. These contextual differences have direct implications for diagnosis and management. Uniform application of platelet thresholds and etiologic assumptions risks misclassification of thrombocytopenia and inappropriate prioritization of investigations. Incorporating regional baseline reference ranges, co-morbidity profiles, and treatment continuity into clinical interpretation can improve diagnostic precision and guide more rational, targeted interventions.

From a clinical perspective, sustained virologic suppression through early and continuous antiretroviral therapy remains the cornerstone of platelet recovery. However, optimal hematologic outcomes require integrated care models that address prevalent co-infections, nutritional deficits, and organ disease that perpetuate thrombocyte dysregulation. Embedding routine platelet monitoring within HIV care pathways and aligning management strategies with regional epidemiology may reduce bleeding risk, mitigate thromboinflammatory complications, and improve long-term outcomes. Future research should prioritize harmonized, multi-regional cohort studies with standardized platelet measurement protocols and systematic adjustment for co-morbid conditions. Such efforts are essential to refine region-sensitive diagnostic thresholds, inform guideline development, and advance more equitable, precision-oriented care for platelet disorders in diverse HIV populations.

Conflicts of Interest

The author declares no conflict of interest

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