International Journal of Advanced Multidisciplinary Research
ISSN: 2393-8870
www.ijarm.com
(A Peer Reviewed, Referred, Indexed and Open Access Journal)
DOI: 10.22192/ijamr Volume 12, Issue 9 -2025

Review Article

DOI: http://dx.doi.org/10.22192/ijamr.2025.12.09.004

Anemia, Immunity, and Infection: Public Health Implications of Hematological Abnormalities in People Living with HIV

***Emmanuel Ifeanyi Obeagu**
Department of Biomedical and Laboratory Science, Africa University, Zimbabwe.
*Corresponding author: Emmanuel Ifeanyi Obeagu, Department of Biomedical and Laboratory Science, Africa University, Zimbabwe, emmanuelobeagu@yahoo.com,
ORCID: 0000-0002-4538-0161

Keywords

HIV,
anemia,
immunity,
hematological abnormalities,
public health

Abstract

Hematological abnormalities are among the earliest and most persistent clinical manifestations of Human Immunodeficiency Virus (HIV) infection, serving as vital indicators of immune suppression, disease progression, and treatment outcomes. Among these, anemia stands out as the most prevalent, affecting up to 80% of individuals with advanced HIV. The interaction between anemia, immunity, and infection forms a complex triad that drives morbidity and mortality in people living with HIV (PLHIV). The pathogenesis of these abnormalities is multifactorial—arising from direct viral effects on bone marrow progenitor cells, chronic inflammation, opportunistic infections, nutritional deficiencies, and antiretroviral drug toxicity.This narrative review explores the intricate interrelationships between anemia, immune dysregulation, and infection in HIV, emphasizing the public health dimensions often overlooked in clinical management. It highlights how hematological abnormalities not only reflect individual disease burden but also mirror systemic public health gaps—such as poverty, undernutrition, limited laboratory infrastructure, and inequitable access to healthcare. From a population health perspective, routine hematologic screening offers a cost-effective strategy for disease monitoring and early intervention in resource-limited settings.Furthermore, the review discusses how integrating hematologic surveillance into HIV care can improve prognostic assessment, inform therapeutic decisions, and reduce treatment-related toxicities. Public health interventions such as nutritional supplementation, infection control, pharmacovigilance, and maternal health programs can significantly mitigate the burden of HIV-associated anemia and cytopenias. Addressing these hematologic complications through a multidisciplinary approach

that combines clinical care, health education, and system strengthening is essential to achieving global HIV targets and improving the quality of life of affected individuals.

Introduction

Human Immunodeficiency Virus (HIV) infection remains one of the most formidable global health challenges, with an estimated 38 million people living with the virus worldwide. Despite remarkable progress in antiretroviral therapy (ART) and the consequent decline in AIDS-related mortality, HIV continues to exert profound systemic effects that extend beyond immunosuppression. Among these, hematological abnormalities represent a major but often under-recognized facet of HIV pathology, reflecting both disease progression and broader public health challenges [1-3].Hematologic complications—including anemia, leukopenia, thrombocytopenia, and pancytopenia—are highly prevalent among individuals with HIV and have significant clinical and epidemiological implications. Anemia, in particular, is the most common hematologic disorder associated with HIV, with prevalence rates ranging from 40% to over 80% depending on disease stage and geographic region. It is closely linked to accelerated disease progression, diminished quality of life, and increased risk of mortality. The etiology of HIV-associated anemia is multifactorial, involving direct viral effects on hematopoietic progenitor cells, chronic immune activation, bone marrow suppression, nutritional deficiencies, and side effects of ART, particularly zidovudine-based regimens [4-7].

The interplay between **anemia, immunity, and infection** in HIV creates a vicious cycle that amplifies disease burden. Immunosuppression predisposes patients to recurrent infections, which in turn promote inflammatory cytokine release, impair erythropoiesis, and worsen anemia. Conversely, anemia itself compromises oxygen delivery to tissues and immune cells, reducing the body's capacity to mount an effective immune response. This interdependence illustrates how hematologic abnormalities are not isolated manifestations but integral components of HIV pathophysiology and progression [8-10].From a public health perspective, hematologic abnormalities among people living with HIV (PLHIV) are important indicators of both individual and community-level health disparities. In low- and middle-income countries (LMICs), the high prevalence of HIV-related anemia often reflects broader determinants such as malnutrition, parasitic coinfections, poor sanitation, and limited access to healthcare services. These systemic issues hinder timely diagnosis and management, exacerbating morbidity and mortality. Moreover, in resource-constrained settings where advanced laboratory tools may not be readily available, basic hematological parameters such as hemoglobin levels, white blood cell counts, and platelet counts serve as cost-effective, accessible, and reliable markers for disease monitoring and treatment outcomes [11-14].

The recognition of hematologic abnormalities as key components of HIV management has profound public health implications. Effective prevention and management require integration across multiple levels of healthcare—linking clinical monitoring with nutritional support, infection control, pharmacovigilance, and community-based education. For example, incorporating routine complete blood count (CBC) testing into HIV care protocols can facilitate early detection of treatment-related toxicities and secondary infections, improving patient outcomes and reducing health system burdens [15-16].Furthermore, addressing the hematologic dimensions of HIV has special relevance for vulnerable populations such as pregnant women, children, and individuals with coinfections like tuberculosis and malaria. Anemia during pregnancy among HIV-positive women increases the risk of preterm birth, low birth weight, and maternal mortality, while in children, it contributes to impaired growth,

neurocognitive delay, and weakened immune defense. Thus, the hematologic complications of HIV are not only clinical manifestations but also indicators of health equity, reflecting the broader intersection between disease biology and social determinants of health [17-20].This review explores the multifaceted relationships between anemia, immunity, and infection in HIV, emphasizing their implications for public health systems and population-level interventions. By integrating clinical, biological, and epidemiological perspectives, the paper highlights the importance of strengthening hematologic surveillance within HIV programs and advancing strategies that bridge laboratory medicine, nutrition, and preventive health. Ultimately, understanding and addressing hematologic abnormalities in HIV is pivotal for improving patient survival, achieving sustainable public health goals, and ensuring holistic care for those affected by the disease.

Hematological Abnormalities in HIV Infection

Hematological abnormalities represent some of the most frequent and clinically significant complications of Human Immunodeficiency Virus (HIV) infection. These alterations reflect both the direct effects of the virus on hematopoietic tissues and the indirect consequences of immune dysregulation, opportunistic infections, nutritional deficiencies, and antiretroviral drug toxicities. The bone marrow, as the central site of blood cell production, is a primary target of HIV-induced injury. Viral proteins, pro-inflammatory cytokines, and secondary infections collectively disrupt hematopoiesis, resulting in a wide spectrum of cytopenias—most notably anemia, leukopenia, and thrombocytopenia [21-22].**Anemia** remains the most prevalent hematologic abnormality in people living with HIV (PLHIV). Its pathogenesis is multifactorial and varies with disease stage and geographic setting. In early infection, anemia may be mild and transient, reflecting chronic inflammation or nutritional deficits. As the disease progresses, the mechanisms become more complex, involving direct HIV invasion of bone marrow progenitor cells, immune-mediated suppression of erythropoiesis, and opportunistic infections that infiltrate or impair marrow function. Chronic activation of inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) inhibits erythropoietin production and iron mobilization, leading to anemia of chronic disease. Moreover, deficiencies of iron, folate, and vitamin B12—common in resource-limited settings—exacerbate red cell production defects [23-24].

Drug-induced anemia is another critical concern in HIV management. Zidovudine, a nucleoside reverse transcriptase inhibitor (NRTI) once widely used in first-line therapy, has been associated with macrocytic anemia due to bone marrow suppression. Similarly, other agents such as ganciclovir and ribavirin may aggravate anemia when used in the treatment of coexisting infections. In women and children, who are more vulnerable to nutritional depletion, the combined effect of infection, inflammation, and medication toxicity often results in severe anemia with serious clinical consequences including fatigue, cognitive impairment, and reduced physical functioning [25-28].**Leukopenia**, particularly **neutropenia**, is another common hematologic manifestation of HIV infection. It arises primarily from decreased bone marrow production, immune-mediated destruction of neutrophils, or drug-related toxicity. Antiretroviral and prophylactic agents such as cotrimoxazole and ganciclovir can depress white blood cell counts, increasing susceptibility to bacterial and fungal infections. Furthermore, direct infection of marrow stromal cells and suppression of colony-stimulating factors contribute to neutropenia, especially in patients with advanced disease or uncontrolled viral replication. Clinically, persistent neutropenia not only predisposes to recurrent infections but also complicates the use of certain myelosuppressive medications, necessitating careful hematologic monitoring during treatment [29].

Thrombocytopenia is another frequent hematologic disturbance seen at various stages of HIV infection. Its mechanisms are multifactorial,

involving immune-mediated platelet destruction, impaired megakaryocyte maturation, and decreased platelet lifespan. In early HIV infection, immune thrombocytopenic purpura (ITP) may occur as a result of autoantibody-mediated platelet destruction. As the disease advances, bone marrow suppression and direct infection of megakaryocytes by HIV further reduce platelet production. Clinically, thrombocytopenia presents with mucocutaneous bleeding, petechiae, and easy bruising, though severe cases may lead to life-threatening hemorrhage. Importantly, successful initiation of ART has been shown to improve platelet counts in most patients, underscoring the reversibility of immune-mediated mechanisms with viral suppression [30].In some individuals, HIV infection leads to **pancytopenia**, a condition characterized by simultaneous reduction in red cells, white cells, and platelets. This severe manifestation often signals bone marrow infiltration by opportunistic pathogens such as *Mycobacterium avium complex*, *Histoplasma capsulatum*, or lymphoma. Pancytopenia is commonly associated with advanced AIDS and indicates profound marrow failure. The clinical implications are grave, including severe immunosuppression, recurrent infections, bleeding tendencies, and poor treatment outcomes [31].

Beyond quantitative cell deficiencies, qualitative hematologic alterations also occur. These include changes in red cell morphology, reduced neutrophil function, and impaired platelet aggregation—all of which further compromise host defense mechanisms. The presence of such abnormalities has important diagnostic and prognostic implications. For instance, declining hemoglobin or platelet counts can serve as early warning signs of treatment failure or disease progression, particularly in settings where viral load testing is unavailable or unaffordable [32].Hematological abnormalities in HIV therefore represent more than laboratory findings—they are dynamic reflections of the interaction between the virus, host immunity, and environmental influences. Their high prevalence across diverse populations underscores the need for integrating hematologic evaluation into routine HIV care. Early detection and management of these abnormalities not only improve quality of life but also enhance treatment outcomes by guiding appropriate therapy adjustments, nutritional support, and infection control measures. Recognizing their public health significance is essential in designing comprehensive HIV care models that address both clinical and systemic determinants of hematologic health (Table 1 [33]).

Table 1: Common Hematological Abnormalities in HIV and Their Pathophysiological Mechanisms

Abnormality	Pathophysiological Mechanism	Clinical Implication
Anemia	Direct viral invasion of progenitor cells, chronic inflammation, nutritional deficiency, ART toxicity (e.g., zidovudine)	Fatigue, impaired immunity, disease progression
Neutropenia	Marrow suppression, immune-mediated destruction, drug-induced toxicity (e.g., ganciclovir, cotrimoxazole)	Increased susceptibility to bacterial and fungal infections
Thrombocytopenia	Immune-mediated platelet destruction, bone marrow suppression, HIV-related megakaryocyte dysfunction	Increased bleeding risk, mucocutaneous hemorrhage
Pancytopenia	Bone marrow infiltration, advanced HIV, opportunistic infections (e.g., Mycobacterium avium complex)	Severe immunosuppression and infection risk

Anemia and Immune Dysfunction in HIV

Anemia is not merely a laboratory finding in HIV infection—it reflects complex immunopathological interactions that influence disease progression and clinical outcomes. The relationship between anemia and immune dysfunction in HIV is deeply interwoven, forming a cyclical pattern in which immunosuppression predisposes to anemia, while anemia itself exacerbates immune impairment. This bidirectional relationship has significant clinical and public health implications, particularly in settings where nutritional deficiencies, opportunistic infections, and limited access to comprehensive care are common.HIV exerts its effects on the hematopoietic system through both direct and indirect mechanisms. Directly, the virus invades bone marrow progenitor cells and stromal elements, impairing their capacity to sustain normal erythropoiesis. Viral proteins such as gp120 and Tat interfere with the growth and differentiation of erythroid precursors, leading to reduced red blood cell production. Indirectly, chronic immune activation—one of the hallmarks of HIV infection—triggers the release of pro-inflammatory cytokines, including interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ). These mediators suppress erythropoietin synthesis in the kidneys, reduce iron availability by increasing hepcidin production, and inhibit erythroid progenitor cell proliferation in the bone marrow. The resulting condition, often termed "anemia of chronic disease," is a common finding in advanced HIV infection [34].

Moreover, immune dysfunction in HIV increases susceptibility to opportunistic infections that further disrupt erythropoiesis. Co-infections such as tuberculosis, malaria, and parvovirus B19 can directly infiltrate or damage the bone marrow, while chronic parasitic infestations contribute to nutritional deficiencies that compound anemia. In malaria-endemic regions, for instance, HIV-infected individuals experience more severe and recurrent malarial anemia due to impaired immune clearance of the parasite. Similarly, coexistent tuberculosis exacerbates systemic inflammation, intensifying anemia through cytokine-mediated iron sequestration and marrow suppression [35].Anemia also feeds back into the immune system, creating a vicious cycle of immune compromise. Reduced hemoglobin levels lead to tissue hypoxia, which impairs the energy-dependent processes of immune cells, including lymphocyte proliferation, phagocytosis, and cytokine production. Oxygen deprivation alters cellular metabolism and weakens both innate and adaptive immune responses, rendering the host more vulnerable to new infections and reactivation of latent pathogens. Additionally, iron metabolism—a central determinant in erythropoiesis—is closely linked to immune function. Dysregulated iron homeostasis in HIV leads to both functional iron deficiency and iron overload, each with deleterious effects: deficiency hampers immune cell activity, while overload promotes oxidative stress and microbial proliferation [36].

The interplay between anemia and immune status in HIV also has prognostic significance. Numerous studies have demonstrated that anemia correlates with lower CD4+ T-cell counts, higher viral loads, and faster progression to AIDS. In fact, hemoglobin concentration is considered an independent predictor of survival among PLHIV, even after adjusting for CD4 count and viral load. The restoration of hemoglobin levels through ART initiation often parallels immune recovery, highlighting the intertwined nature of hematologic and immune reconstitution [37].From a therapeutic perspective, addressing anemia in HIV requires a multifaceted approach. Effective viral suppression through ART remains the cornerstone, as it reduces inflammation, restores marrow function, and reverses immune-mediated suppression of erythropoiesis. Nutritional interventions, including iron, folate, and vitamin B12 supplementation, are essential adjuncts, especially in regions where dietary insufficiency and malabsorption are prevalent. In selected cases, erythropoiesis-stimulating agents (ESAs) may be beneficial, though their use must be balanced against cost and potential side effects. Equally important is the identification and treatment of opportunistic infections that

exacerbate anemia, such as malaria, tuberculosis, and chronic bacterial infections [37].

From a public health viewpoint, the coexistence of anemia and immune dysfunction among PLHIV highlights the interconnectedness of infectious disease control, nutrition, and health system capacity. Routine screening for anemia and immune status can serve as a low-cost, high-impact tool for early identification of patients at risk of rapid disease progression. Integrating hematologic and immunologic assessments into HIV care not only improves individual patient outcomes but also contributes to the broader goal of achieving sustained viral suppression and reducing population-level morbidity [38].

Infection, ART, and Hematologic Toxicity

The hematologic landscape in HIV infection is shaped not only by the virus itself but also by a complex interplay of opportunistic infections and the effects of antiretroviral therapy (ART). While ART has dramatically improved survival and reduced HIV-associated morbidity, its use is not without adverse hematologic consequences. Opportunistic infections and pharmacologic toxicity together represent critical determinants of blood cell abnormalities in people living with HIV (PLHIV), influencing both clinical outcomes and quality of life [38].Opportunistic infections remain a major cause of hematologic disruption in HIV, particularly in individuals with advanced immunosuppression. Infections such as *Mycobacterium tuberculosis*, *Mycobacterium avium complex* (MAC), *Histoplasma capsulatum*, and *Cryptococcus neoformans* can infiltrate the bone marrow, leading to direct suppression of hematopoiesis and pancytopenia. These pathogens not only destroy marrow architecture but also induce chronic inflammatory responses that disrupt normal red and white blood cell production. Parvovirus B19, for instance, selectively infects erythroid progenitor cells, causing pure red cell aplasia that manifests as severe refractory anemia. Similarly, chronic viral co-infections such as hepatitis B and C contribute to marrow suppression and thrombocytopenia through immune-mediated and fibrotic mechanisms [39].

In malaria-endemic regions, coinfection with *Plasmodium* species further aggravates hematologic impairment. Malaria-induced hemolysis, in combination with HIV-associated bone marrow dysfunction, results in profound anemia that is often resistant to standard treatment. The coexistence of these infections highlights the public health challenges of managing HIV in resource-limited settings, where multiple endemic pathogens coexist and exacerbate hematologic morbidity [40].The introduction of ART revolutionized HIV care, transforming it from a fatal disease into a manageable chronic condition. However, antiretroviral drugs themselves may cause or worsen hematologic toxicity. Among the earliest and most recognized agents associated with bone marrow suppression is zidovudine (AZT), a nucleoside reverse transcriptase inhibitor (NRTI). Zidovudine-induced macrocytic anemia results from mitochondrial toxicity and impaired DNA synthesis in erythroid progenitor cells. This form of anemia is often dose-dependent and may necessitate drug substitution when severe. Although the widespread transition to newer ART regimens has reduced zidovudine use, it remains a common component of first-line therapy in many low-income countries due to cost and availability, perpetuating the risk of drug-induced cytopenias [41].

Other antiretroviral agents may also contribute to hematologic disturbances through indirect mechanisms. For instance, protease inhibitors have been implicated in the development of insulin resistance and metabolic changes that alter bone marrow microenvironmental function. Non-nucleoside reverse transcriptase inhibitors (NNRTIs), such as nevirapine, can cause hypersensitivity reactions and immune-mediated cytopenias. Additionally, pharmacologic interactions between ART and medications used to treat opportunistic infections—such as ganciclovir, cotrimoxazole, and rifampicin—can amplify myelosuppression. This overlap of drug toxicity and disease-related bone marrow

suppression poses significant clinical challenges, especially in patients with advanced HIV disease who require multiple concurrent therapies [42].The pathophysiology of ART-related hematologic toxicity extends beyond marrow suppression. Mitochondrial dysfunction plays a key role in the development of anemia, neutropenia, and thrombocytopenia. Antiretroviral drugs that impair mitochondrial DNA polymerase γ lead to defective oxidative phosphorylation, increased reactive oxygen species production, and cellular apoptosis within hematopoietic progenitors. Over time, these processes result in reduced cell proliferation and the accumulation of cytopenias, particularly when compounded by nutritional deficiencies and ongoing immune activation [43].

Clinically, hematologic toxicities manifest across a spectrum ranging from mild asymptomatic cytopenias to life-threatening marrow failure. The presentation may vary depending on the drug regimen, duration of exposure, and coexisting infections. Patients on zidovudine may develop progressive fatigue and pallor due to anemia, whereas ganciclovir-induced neutropenia presents with recurrent bacterial or fungal infections. Thrombocytopenia may lead to mucosal bleeding, purpura, and, in severe cases, intracranial hemorrhage. These manifestations not only complicate HIV management but also threaten adherence to ART, as patients may discontinue or alter regimens due to perceived drug side effects [44].From a public health perspective, the intersection of infection and ART toxicity underscores the need for **comprehensive hematologic surveillance** within HIV programs. Routine complete blood count (CBC) monitoring enables early detection of drug-induced cytopenias, facilitating timely therapeutic adjustments before complications arise. In resource-limited settings where laboratory infrastructure is inadequate, point-of-care diagnostic tools and simplified screening algorithms could serve as effective alternatives. Pharmacovigilance systems must also be strengthened to monitor the safety of ART and other drugs used in HIV care [45].Preventive strategies are equally important. Optimizing ART regimens to minimize marrow toxicity, ensuring adequate nutrition, and preventing or promptly treating opportunistic infections can significantly reduce hematologic complications. Transitioning from older NRTIs like zidovudine to newer agents such as tenofovir and dolutegravir has been shown to lower the incidence of anemia and neutropenia. Education and training for healthcare workers in recognizing early signs of drug toxicity and infection-related cytopenias are vital components of sustainable HIV care delivery.

Public Health Implications

The hematological abnormalities associated with HIV infection extend beyond clinical pathology, presenting profound public health challenges that influence disease management, healthcare delivery, and population well-being. These abnormalities—particularly anemia, leukopenia, and thrombocytopenia—serve as critical indicators of both individual immune status and systemic weaknesses within healthcare systems. In many low- and middle-income countries (LMICs), where HIV prevalence remains high, hematologic complications compound existing public health burdens by increasing vulnerability to opportunistic infections, impairing productivity, and escalating healthcare costs [46].Anemia, for instance, reduces physical capacity and cognitive function, limiting the ability of affected individuals to maintain employment and adhere to antiretroviral therapy (ART). This contributes to socioeconomic instability and perpetuates cycles of poverty and illness. Moreover, anemia in HIV-positive pregnant women is strongly associated with adverse maternal and neonatal outcomes, including preterm birth, low birth weight, and increased maternal mortality—an intersection that underscores the importance of integrating HIV and reproductive health programs [47].

From a surveillance perspective, hematological parameters offer valuable, cost-effective biomarkers for monitoring disease progression and treatment efficacy in resource-constrained settings. Routine hemoglobin, white cell, and platelet counts can serve as proxy indicators for

immune function where access to CD4 and viral load testing is limited. This diagnostic integration strengthens public health monitoring systems and enhances early detection of ART toxicity or treatment failure [48].However, the persistence of hematologic disorders among people living with HIV also reflects structural inequities—such as food insecurity, poor sanitation, limited access to micronutrient supplementation, and inadequate pharmacovigilance. Addressing these determinants requires a holistic approach that transcends biomedical care. Public health strategies should focus on community-based nutritional programs, infection control measures, and education campaigns that promote adherence to ART while reducing the risk of anemia-inducing infections like malaria and tuberculosis [49].Furthermore, strengthening laboratory capacity for hematologic testing and drug safety monitoring is essential for sustainable HIV management. Policymakers must prioritize integrating hematologic assessments into national HIV guidelines, ensuring equitable access to diagnostic services, and fostering interdisciplinary collaborations between hematologists, infectious disease specialists, and public health practitioners (Table 2).

Table 2: Public Health Strategies for Managing Hematologic Abnormalities in HIV

Strategy	Public Health Approach	Expected Outcome
Routine Hematologic Screening	Integration of CBC tests into HIV care protocols	Early detection of anemia, neutropenia, and thrombocytopenia
Nutrition and Micronutrient Support	Iron and folate supplementation, fortified food programs	Reduced anemia prevalence and improved immunity
Pharmacovigilance and ART Optimization	Monitoring and switching from marrow-toxic drugs	Decreased drug-induced cytopenias
Community Health Education	Promoting awareness of anemia symptoms and nutrition	Improved health-seeking behavior
Health System Strengthening	Equipping primary facilities for hematology testing	Enhanced diagnostic capacity and continuity of care

Conclusion

Hematological abnormalities remain central to the clinical and public health landscape of HIV infection. Anemia, leukopenia, and thrombocytopenia are not only reflections of viral pathogenicity and immune compromise but also indicators of broader systemic challenges—including malnutrition, coinfections, and healthcare inequities. These hematologic disturbances profoundly influence disease progression, treatment adherence, and survival outcomes among people living with HIV.The interplay between anemia, immunity, and infection highlights the need for an integrated approach that combines clinical management with public health interventions. Routine hematologic monitoring, coupled with nutritional support, infection control, and pharmacovigilance, should be prioritized as part of comprehensive HIV care. Strengthening laboratory capacity, improving ART safety monitoring, and expanding access to affordable diagnostic tools can enhance early detection and timely management of hematologic complications.At the population level, addressing these challenges requires robust policies that link HIV programs with maternal health, nutrition, and primary care services. A multidisciplinary approach—bridging hematology, infectious disease, and public health—will be vital to reducing morbidity and improving quality of life in affected populations. Ultimately, mitigating the burden of HIV-associated hematological abnormalities is not only a clinical necessity but also a cornerstone of achieving global HIV control and health equity goals.

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DOI:10.22192/ijamr.2025.12.09.004	

How to cite this article:
Emmanuel Ifeanyi Obeagu. (2025). Anemia, Immunity, and Infection: Public Health Implications of Hematological Abnormalities in People Living with HIV. Int. J. Adv. Multidiscip. Res. 12(9): 28-39.
DOI: http://dx.doi.org/10.22192/ijamr.2025.12.09.004