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HIV-Induced Anemia: Mechanisms, Diagnosis, and Clinical Management

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Abstract

Anemia is a common and clinically significant complication of HIV infection, with prevalence rates varying widely based on disease stage and treatment access. It is associated with impaired quality of life, faster HIV progression, and increased mortality. Despite the success of antiretroviral therapy (ART) in controlling viral replication, anemia remains prevalent, especially in resource-limited settings. The etiology of HIV-induced anemia is multifactorial, involving direct effects of the virus on bone marrow progenitor cells, chronic inflammation, opportunistic infections, nutritional deficiencies, and adverse effects of medications. The underlying pathophysiology includes impaired erythropoiesis due to viral suppression of bone marrow function, dysregulated cytokine production, and iron sequestration mediated by hepcidin. Additional contributors such as vitamin B12 and folate deficiencies, as well as ART-associated myelotoxicity—particularly from zidovudine—further compound the risk. Clinical presentation ranges from asymptomatic cases to severe fatigue and cardiovascular compromise. Diagnostic approaches rely on a thorough evaluation of red cell indices, reticulocyte response, iron status, vitamin levels, and bone marrow function. Management involves addressing the underlying cause, optimizing ART regimens, correcting nutritional deficits, and using erythropoiesis-stimulating agents or transfusions when necessary. Early recognition and tailored treatment are vital for improving hematologic outcomes and overall prognosis. Given the complex interplay between HIV pathogenesis and anemia, integrated clinical strategies and equitable access to diagnostics and therapeutics are essential in reducing the burden of this condition globally.

Keywords: *HIV, Anemia, Hematopoiesis, Antiretroviral Therapy, Inflammation*

Introduction

Anemia remains one of the most frequent hematologic complications in individuals living with human immunodeficiency virus (HIV), affecting both adults and children across all stages of the disease. It is often the earliest and most persistent abnormality observed in HIV-infected patients and is associated with a decline in physical functioning, reduced quality of life, impaired immune response, and increased mortality. As the global burden of HIV persists—particularly in sub-Saharan Africa and low-resource settings—anemia continues to be a pressing clinical concern despite the widespread implementation of antiretroviral therapy (ART) [1-5].The prevalence of anemia in people living with HIV varies from 20% to over 80%, depending on factors such as disease stage, viral load, nutritional status, co-infections, and ART use. It is most prevalent among individuals with advanced immunosuppression or those with comorbid opportunistic infections. Anemia is not merely a marker of disease severity but also an independent predictor of HIV progression and poor treatment outcomes. The presence of anemia has been shown to negatively impact adherence to ART, increase the risk of hospitalization, and contribute to early mortality [6-9].HIV-associated anemia is a complex, multifactorial condition. It results from a combination of direct viral effects on bone marrow progenitor cells, immune-mediated suppression of hematopoiesis, nutrient deficiencies, medication-related toxicity, and complications from opportunistic infections and malignancies. The clinical manifestation of anemia in HIV may vary in severity and etiology, requiring careful evaluation to identify and manage the underlying cause. Importantly, the persistence of anemia despite viral suppression in some individuals highlights the need for targeted diagnostic and therapeutic strategies [10-12].

At the core of HIV-induced anemia lies the suppression of erythropoiesis. HIV has been shown to disrupt the bone marrow microenvironment, impairing the proliferation and differentiation of erythroid precursors. Additionally, chronic immune activation and systemic inflammation drive the production of inhibitory cytokines such as tumor necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ), and interleukin-6 (IL-6), which contribute to anemia of chronic disease through reduced erythropoietin responsiveness and functional iron deficiency mediated by hepcidin upregulation [13].Nutritional deficiencies further exacerbate anemia in HIV-infected individuals. In many cases, patients experience deficiencies in iron, folate, or vitamin B12 due to malabsorption, poor dietary intake, or gastrointestinal infections. These deficiencies impair DNA synthesis in erythroblasts, leading to ineffective erythropoiesis and megaloblastic changes. Additionally, gastrointestinal blood loss caused by opportunistic infections or neoplasms may contribute to chronic blood loss and iron depletion, particularly in advanced disease stages [14].Pharmacologic factors also play a significant role in the development and persistence of anemia. Zidovudine (AZT), one of the earliest nucleoside reverse transcriptase inhibitors (NRTIs), is well known for its myelotoxic effects, leading to macrocytic anemia in many patients. Other medications used in HIV management, such as cotrimoxazole, ganciclovir, and certain chemotherapeutic agents for AIDS-related malignancies, may contribute to bone marrow suppression or hemolysis. Identifying and managing drug-induced anemia is therefore a critical component of comprehensive HIV care [15-18].

The development of anemia in individuals with HIV infection is driven by a multifactorial pathophysiologic process that involves direct viral effects on hematopoietic tissues, chronic immune activation, cytokine dysregulation, impaired erythropoietin production and response, nutritional deficiencies, and drug-induced myelosuppression[19]. These mechanisms may act independently or synergistically, contributing to both acute and chronic anemic states (Table 1).

Table 1: Pathophysiology of HIV-Induced Anemia

Mechanism	Description	Clinical Implications
Direct HIV Bone Marrow Suppression	HIV infects stromal and progenitor cells, impairing erythropoiesis and marrow cellularity.	Normocytic anemia; associated with low reticulocyte count and pancytopenia in advanced cases.
Chronic Inflammation & Cytokine Release	Elevated TNF-α, IL-1, and IFN-γ suppress erythropoietin response and iron utilization.	Anemia of chronic disease with normocytic, normochromic features; low serum iron but normal/high ferritin.
Opportunistic Infections	Infections such as parvovirus B19, CMV, TB, and fungal pathogens infiltrate or suppress marrow.	Severe or aplastic anemia; may be reversible with infection control.
Nutritional Deficiencies	Common deficiencies include iron, folate, and vitamin B12 due to malabsorption or poor intake.	Microcytic (iron deficiency) or macrocytic (B12/folate deficiency) anemia.
ART-Related Myelotoxicity	Drugs like zidovudine (AZT) inhibit DNA synthesis in erythroid precursors.	Macrocytic anemia; dose-dependent; may resolve with drug substitution.
Autoimmune Hemolysis	HIV-related immune dysregulation may trigger hemolytic anemia or pure red cell aplasia.	Elevated LDH, indirect bilirubin; low haptoglobin; positive Coombs test.
Renal Insufficiency	HIV-associated nephropathy reduces erythropoietin production.	Normocytic anemia with low reticulocyte count; often requires ESA therapy.

1. Direct HIV-Mediated Bone Marrow Suppression

HIV can indirectly impair erythropoiesis by infecting the bone marrow microenvironment, particularly stromal cells and macrophages. Although hematopoietic progenitor cells (CD34+ cells) are not efficiently infected by HIV, the altered stromal support and release of toxic viral proteins such as gp120 and Tat can lead to apoptosis and impaired proliferation of erythroid precursors. Histologic examination of bone marrow from HIV-infected individuals often shows hypocellularity, dysplasia, or erythroid hypoplasia [20-21].

2. Chronic Inflammation and Cytokine Dysregulation

Chronic immune activation is a hallmark of HIV infection, even during effective antiretroviral therapy. Elevated levels of inflammatory cytokines—such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ), and interleukin-6 (IL-6)—suppress erythropoiesis through multiple mechanisms. These cytokines inhibit erythroid progenitor proliferation, shorten red blood cell lifespan, and decrease bone marrow sensitivity to erythropoietin. Additionally, IL-6 induces the hepatic production of hepcidin, a peptide hormone that inhibits intestinal iron absorption and traps iron in macrophages, leading to functional iron deficiency despite adequate iron stores [22-24].

3. Erythropoietin Deficiency and Resistance

Erythropoietin (EPO), primarily produced by the kidneys, is a crucial driver of red blood cell production. In advanced HIV infection, endogenous EPO levels may be inappropriately low for the degree of anemia, partly due to renal dysfunction or cytokine-mediated suppression of EPO synthesis. Moreover, the inflammatory environment in HIV contributes to resistance to EPO, as inflammatory cytokines downregulate EPO receptor expression and signaling in erythroid progenitors [25-26].

4. Nutritional Deficiencies

HIV-infected individuals frequently suffer from deficiencies in iron, folate, and vitamin B12, all of which are essential for normal erythropoiesis. Malnutrition, gastrointestinal malabsorption, chronic diarrhea, and concurrent infections such as Giardia or cytomegalovirus colitis contribute to micronutrient losses. Iron deficiency results in microcytic, hypochromic anemia, while folate and vitamin B12 deficiencies produce macrocytic anemia with megaloblastic marrow changes. These nutritional deficits are especially common in low-income settings and may coexist with anemia of chronic disease [27].

5. Bone Marrow Infiltration and Opportunistic Infections

Opportunistic infections, such as Mycobacterium tuberculosis, Mycobacterium avium complex (MAC), and parvovirus B19, can infiltrate or infect the bone marrow and directly suppress erythropoiesis. Parvovirus B19 is particularly notable for causing pure red cell aplasia by infecting erythroid progenitor cells, leading to a profound reticulocytopenia. Similarly, HIV-associated lymphomas or metastatic cancers can infiltrate bone marrow spaces, displacing normal hematopoiesis [28].

6. Hemolysis and Immune-Mediated Red Cell Destruction

In some HIV-infected individuals, hemolytic anemia may occur due to autoimmune processes or drug-induced hemolysis. Autoimmune hemolytic anemia (AIHA) is more frequent in advanced disease or in association with other autoimmune conditions. Drug-induced hemolysis can occur with medications such as dapsone or sulfonamides, especially in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency. Hemolysis contributes to the overall burden of anemia and may require targeted therapy [29].

7. Antiretroviral and Another Drug Toxicity

Several ART drugs, particularly zidovudine (AZT), are associated with myelosuppression. Zidovudine-induced anemia is typically macrocytic and dose-dependent, resulting from inhibition of DNA synthesis in rapidly dividing erythroid cells. Additional drugs commonly used in HIV management, such as ganciclovir, trimethoprim-sulfamethoxazole, and chemotherapy agents, also contribute to bone marrow suppression or hemolysis, exacerbating anemia [30].

Clinical Presentation

The clinical manifestations of anemia in people living with HIV (PLHIV) are variable and depend largely on the severity, duration, and underlying cause of the anemia. Mild anemia may be asymptomatic and only detected through routine laboratory screening. However, as the hemoglobin level declines, patients may present with symptoms including fatigue, weakness, pallor, shortness of breath, dizziness, palpitations, and reduced exercise tolerance. In more severe cases, there may be signs of high-output cardiac failure, such as tachycardia, orthopnea, or peripheral edema.HIV-related anemia often coexists with systemic manifestations of the underlying infection or comorbidities. For instance, patients with advanced disease may present with constitutional symptoms such as weight loss, night sweats, and fever, which can obscure the diagnosis of anemia. In children, growth retardation, poor school performance, and irritability may be additional signs. Symptoms related to specific causes, such as glossitis and neuropathy in vitamin B12 deficiency or jaundice in hemolysis, may also guide further diagnostic evaluation.The pattern of anemia—whether microcytic, normocytic, or macrocytic—can provide clues about the etiology. Microcytic anemia is suggestive of iron deficiency or chronic blood loss, normocytic anemia is typical of anemia of chronic disease or renal insufficiency, and macrocytic anemia is commonly associated with zidovudine therapy or vitamin B12/folate deficiency. A comprehensive diagnostic approach is therefore critical in determining the underlying cause and tailoring management appropriately [28-29].

Diagnostic Evaluation

The diagnosis of HIV-induced anemia requires a systematic evaluation beginning with a complete blood count (CBC), which provides essential information about hemoglobin concentration, hematocrit, red cell indices (MCV, MCH, MCHC), and reticulocyte count. The mean corpuscular volume (MCV) is particularly useful in narrowing down potential causes. Reticulocyte count helps assess bone marrow response; a low reticulocyte count suggests decreased production, while an elevated count may point to hemolysis or blood loss.Iron studies, including serum iron, ferritin, transferrin saturation, and total iron-binding capacity (TIBC), are vital for assessing iron status. However, ferritin is an acute-phase reactant and may be elevated in inflammation, masking true iron deficiency. Soluble transferrin receptor (sTfR) levels or the sTfR/log ferritin index may provide better accuracy in distinguishing iron deficiency from anemia of chronic disease. Measurement of serum vitamin B12 and folate is also warranted, particularly in patients with macrocytic anemia.Other important tests include serum erythropoietin levels (if renal impairment is suspected), lactate dehydrogenase (LDH), haptoglobin, and a peripheral blood smear to evaluate for hemolysis. Bone marrow aspiration and biopsy may be indicated in cases of unexplained anemia, persistent cytopenias, or suspected marrow infiltration by infections or malignancies. Additional investigations may be needed to identify contributing factors such as opportunistic infections (e.g., parvovirus B19 PCR, tuberculosis screening), drug toxicities, or nutritional deficiencies.A thorough medication history is essential to identify potential myelotoxic agents or hemolytic triggers. Co-infections with hepatitis B or C, which can also contribute to hematologic abnormalities, should be screened in patients presenting with anemia. Finally, staging the severity of anemia using the World Health Organization (WHO) criteria can aid in assessing urgency and guiding management decisions [28-30].

Clinical Management

The management of HIV-induced anemia requires a comprehensive and individualized approach aimed at identifying and correcting the underlying cause, alleviating symptoms, and improving hematologic outcomes. Effective treatment not only improves quality of life but may also reduce HIV-related morbidity and mortality. The cornerstone of management involves optimizing antiretroviral therapy (ART), correcting nutritional deficiencies, treating coexisting infections, and, when indicated, providing supportive care such as erythropoiesis-stimulating agents (ESAs) or blood transfusions [31].

1. Optimization of Antiretroviral Therapy (ART)

In many cases, effective suppression of HIV through ART leads to gradual improvement in anemia. ART reduces viral replication, decreases immune activation, and helps restore bone marrow function. However, specific ART agents—especially zidovudine (AZT)—are associated with myelotoxicity and macrocytic anemia. Substituting AZT with less toxic agents (e.g., tenofovir or abacavir) often results in hematologic improvement. Monitoring hemoglobin levels and switching ART regimens when drug-induced anemia is suspected are essential steps in management [32].

2. Treatment of Nutritional Deficiencies

Iron, folate, and vitamin B12 deficiencies are common contributors to anemia in people living with HIV. Oral or parenteral iron supplementation should be provided in cases of confirmed iron deficiency. Careful consideration is needed to avoid indiscriminate iron supplementation in patients with anemia of chronic disease, where iron sequestration rather than absolute deficiency is the primary issue. Folate and vitamin B12 should be replenished based on serum levels or clinical suspicion. Nutritional counseling and food support programs are especially important in resource-limited settings [33].

3. Management of Co-Infections and Bone Marrow Suppression

Opportunistic infections such as tuberculosis, cytomegalovirus, and parvovirus B19 can cause or exacerbate anemia. Appropriate antimicrobial treatment, such as anti-TB therapy or intravenous immunoglobulin for parvovirus-induced pure red cell aplasia, is necessary for hematologic recovery. In cases of bone marrow infiltration or HIV-related malignancies, specific oncologic or hematologic interventions may be needed, including chemotherapy, antiretroviral optimization, or supportive transfusions [34].

4. Use of Erythropoiesis-Stimulating Agents (ESAs)

Recombinant human erythropoietin (rHuEPO) can be beneficial in select HIV-infected patients with anemia, especially when endogenous EPO levels are inappropriately low. Studies have shown improved hemoglobin levels and quality of life with ESA use in patients receiving zidovudine. However, ESAs should be used judiciously, considering potential risks such as thromboembolic events and hypertension. Regular monitoring of hemoglobin response and iron status is recommended during ESA therapy [35].

5. Blood Transfusion and Supportive Care

Blood transfusion may be required in cases of severe or symptomatic anemia, particularly when hemoglobin levels fall below 7–8 g/dL or when there is evidence of cardiovascular compromise. While transfusions provide immediate symptomatic relief, they do not address the underlying cause and carry risks such as alloimmunization, iron overload, and transfusion-transmitted infections. Transfusion should be reserved for acute management or when other interventions have failed or are contraindicated [35].

6. Monitoring and Follow-Up

Ongoing monitoring of hemoglobin levels, red cell indices, reticulocyte response, and iron parameters is essential for evaluating treatment efficacy and guiding adjustments. Regular follow-up visits should include review of ART adherence, nutritional status, and screening for new infections or drug toxicity. In pediatric populations, growth monitoring and developmental assessment are also important. Longitudinal care with a multidisciplinary approach—including infectious disease specialists, hematologists, and nutritionists—enhances outcomes and patient satisfaction.

Prognosis and Public Health Considerations

Prognosis

Anemia in people living with HIV (PLHIV) is more than a hematologic abnormality—it is a marker of disease progression and a predictor of poor clinical outcomes. Numerous studies have shown that anemia is independently associated with increased morbidity, reduced quality of life, and higher mortality, particularly in individuals with advanced immunosuppression and low CD4+ T-cell counts. The severity and persistence of anemia strongly influence overall prognosis, especially when left untreated or when multifactorial contributors are not addressed comprehensively.Timely diagnosis and effective management of anemia can significantly improve the prognosis of HIV-infected individuals. Restoration of normal hemoglobin levels is associated with better functional status, cognitive performance, and immune recovery following ART initiation. In pediatric populations, correction of anemia supports better neurodevelopmental outcomes and growth. Prognosis is also influenced by access to care, nutritional status, comorbidities, and adherence to ART. The availability of safer ART regimens and improved screening tools for anemia have enhanced outcomes over the past two decades, but disparities persist in resource-limited settings [34].

Public Health Considerations

From a public health perspective, HIV-induced anemia represents both a clinical challenge and an opportunity for system-wide interventions. Anemia prevalence among HIV-infected individuals ranges from 20% to over 70%, depending on region, disease stage, ART access, and nutritional context.

In sub-Saharan Africa and other low-income regions, the burden is amplified by widespread nutritional deficiencies, coinfections such as malaria and tuberculosis, and limited diagnostic infrastructure. Integrating routine anemia screening into HIV care platforms is crucial for early detection and intervention.Scaling up access to ART remains the most effective public health strategy to reduce anemia prevalence and improve survival. However, policy frameworks must also prioritize food security, micronutrient supplementation, and management of coinfections. Task-shifting to community health workers, decentralizing care, and leveraging digital tools for anemia monitoring can extend services to underserved populations. Additionally, maternal HIV infection compounded by anemia increases the risk of adverse pregnancy outcomes and vertical HIV transmission, necessitating enhanced antenatal screening and care.Research and surveillance programs must focus on understanding region-specific anemia etiologies in PLHIV, optimizing diagnostic algorithms, and evaluating cost-effective interventions. Public health campaigns that link anemia control with HIV management—particularly in pediatric, adolescent, and pregnant populations—are essential for holistic care delivery. Ultimately, addressing HIV-induced anemia contributes directly to achieving broader health goals, including the UNAIDS 95-95-95 targets and the Sustainable Development Goals (SDGs) on health and well-being [35].

Conclusion

Anemia remains a prevalent and clinically significant complication in individuals living with HIV, affecting disease progression, treatment outcomes, and quality of life. The pathogenesis of HIV-induced anemia is complex and multifactorial, involving direct viral effects on hematopoiesis, chronic inflammation, nutritional deficiencies, opportunistic infections, and drug toxicity. A comprehensive understanding of these mechanisms is critical for effective diagnosis and targeted interventions.Prompt identification and appropriate management of anemia—including optimization of ART, correction of nutritional deficiencies, treatment of co-infections, and judicious use of erythropoietin or transfusions—can substantially improve clinical outcomes. Equally important is routine monitoring and individualized care that addresses the specific etiologies of anemia in diverse populations, including vulnerable groups such as children and pregnant women.

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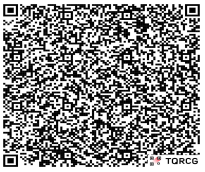	Website: www.ijcrcps.com
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