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Medical Science

To Cite:
Radziwonka A. Hematological signs of early Human Immunodeficiency Virus infection. *Medical Science* 2025; 29: e103ms3629
doi: https://doi.org/10.54905/disssi.v29i161.e103ms3629

Authors' Affiliation:
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

***Corresponding author**
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland; https://orcid.org/0009-0009-0371-474X
Email: ag.radziwonka@gmail.com;

Peer-Review History
Received: 21 March 2025
Reviewed & Revised: 29/March2025 to 27/June/2025
Accepted: 03 July 2025
Published: 11 July 2025

Peer-review Method
External peer-review was done through double-blind method.

Medical Science
pISSN 2321–7359; eISSN 2321–7367

Hematological signs of early Human Immunodeficiency Virus infection

Agnieszka Radziwonka*

ABSTRACT

Introduction: During early HIV (Human Immunodeficiency Virus) infection many individuals develop nonspecific, flu-like symptoms, including fever, lymphadenopathy, rash, and malaise, although a significant proportion of the patients remain asymptomatic. In the acute phase, a high plasma viral load and a transient decline in CD4+ T lymphocytes contribute to increased infectiousness. Early diagnosis of HIV infection is critical, as prompt initiation of antiretroviral therapy during this period can limit immune system damage and reduce transmission risk. This narrative review was based on a comprehensive analysis of the literature to summarize current insights into the epidemiology, pathogenesis, and laboratory diagnosis of HIV-infected patients in the early stages. *Aim*: This article explores the frequency and clinical impact of blood-related changes during the initial phase of HIV infection, with particular attention to anemia, reductions in white blood cells, and low platelet counts as early diagnostic indicators. *State of Knowledge*: Hematological abnormalities are common in early HIV infection, especially anemia, leukopenia, and thrombocytopenia. These abnormalities arise from factors such as immune suppression marked by low CD4+ T-cell counts, disease stage, and co-infections. Their prevalence tends to decrease following initiation of highly active antiretroviral therapy (HAART). The exact mechanism underlying hematopoietic dysfunction in early HIV remains incompletely understood. *Conclusions*: Blood cell irregularities often serve as key early signs of HIV infection and disease advancement. This article demonstrates how standard blood work can identify HIV-related changes, emphasizing that unexplained low blood counts should prompt HIV testing as part of the diagnostic workup.

Keywords: Human Immunodeficiency Virus, Lymphopenia, Anemia, Leukopenia, Hematology

1. INTRODUCTION

Hematological disturbances frequently occur during the early stages of HIV infection, impacting red blood cells, platelets, and white blood cells. Among these, anemia is the most prevalent, typically manifesting as normocytic, normochromic anemia, which results from chronic inflammation, direct viral inhibition of erythropoiesis, and opportunistic infections (Damtie et al., 2021).

Leukopenia is frequently observed in HIV infection, and the factors that cause it are immune cell destruction and bone marrow suppression. This condition increases susceptibility to infections, and its occurrence can differ depending on whether antiretroviral therapy has been started or not (Shi et al., 2014).

Anemia is the most frequent hematological abnormality in patients with HIV. It reduces the quality of life and elevates mortality. In most cases, patients with HIV exhibit normocytic and normochromic anemia. It is also associated with low reticulocyte levels (De Santis et al., 2011).

Neutropenia is common among patients infected with HIV. It affects approximately 10% to 50% of them (Levine et al., 2006). The progression of HIV disease frequently causes neutropenia and compromises immunological function via diverse mechanisms. Profound decreases in neutrophil numbers correlate with worse clinical outcomes (Li et al., 2025).

Low platelet counts often appear in the early stages of infection. Thrombocytopenia occurs mainly due to immune system destruction of platelets and poor platelet production. Starting antiretroviral treatment usually helps both low red blood cells and platelets recover, though some patients may still struggle with leukopenia (Tilahun et al., 2022). These hematological changes correlate with declining CD4+ T-cell counts and elevated viral loads and also serve as important markers for disease progression and treatment monitoring (Suja et al., 2020). Regular hematological evaluation is crucial to guide the clinical management of early HIV infection. Some people do not have any symptoms after acquiring HIV, which leads them to be unaware of it (Apoola et al., 2002).

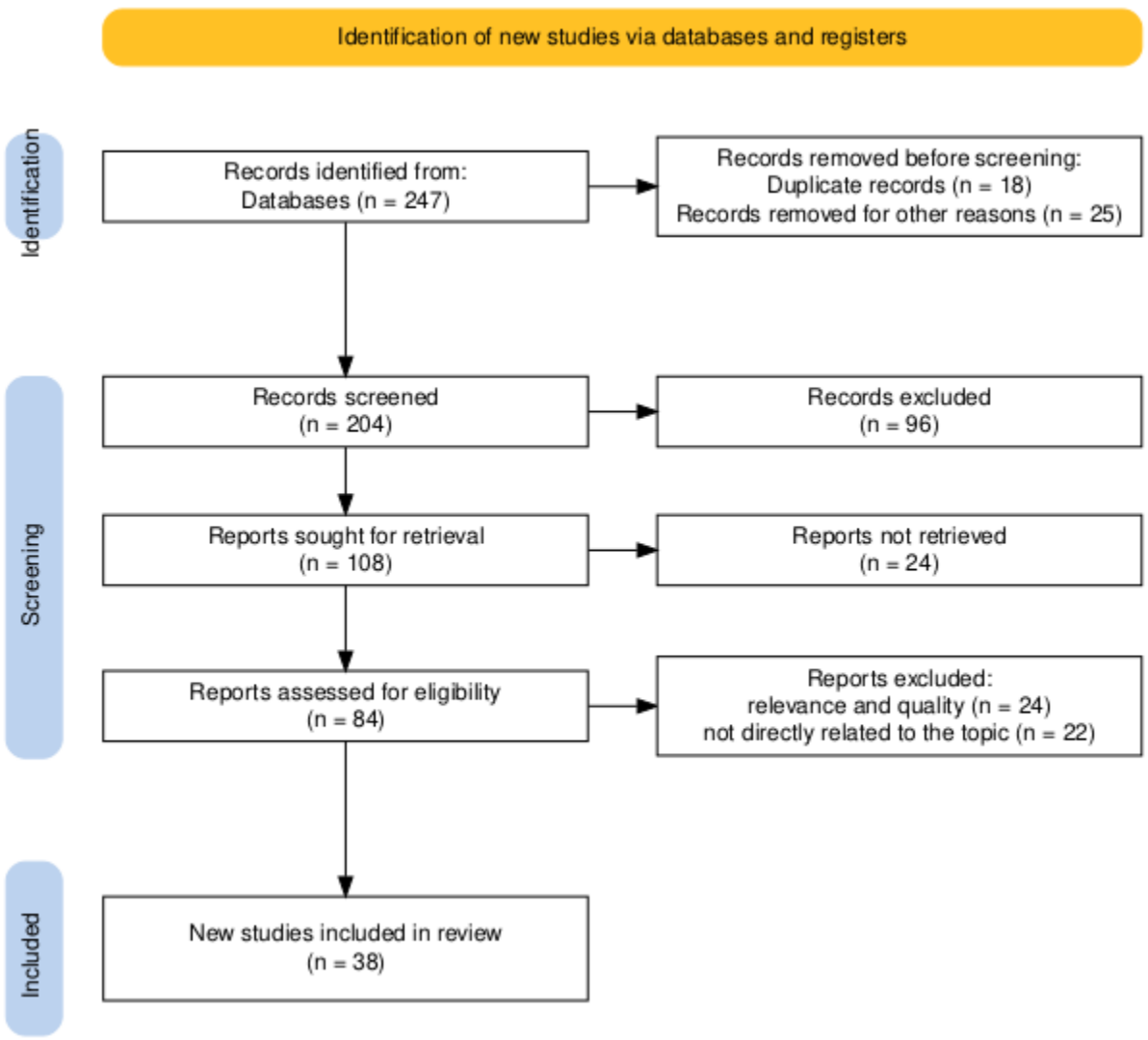

Fig 1: PRISMA consort chart of selected studies

2. REVIEW METHODS

We conducted the research using the PubMed database to identify relevant publications from January 1998 to February 2025. We employed the following keywords in various combinations: ("HIV" OR "human immunodeficiency virus") AND ("hematological signs" OR "hematologic abnormalities" OR "anemia" OR "cytopenia") AND ("early infection" OR "acute infection" OR "primary infection"). In the review we included original research studies, systematic reviews, meta-analyses, and randomized controlled trials. We conducted the study following the PRISMA standards (Figure 1). We screened 204 articles, retrieved a broad set of 108 publications, and assessed 84 of them. We excluded 24 articles, case reports, and meta-analyses focusing exclusively on chronic or late-stage HIV infection. To maintain specificity, we excluded 22 articles not directly related to the topic. After screening for relevance and quality, we selected thirty-eight articles for inclusion.

3. RESULTS & DISCUSSION

Definition, Characteristics, and Epidemiology

The initial clinical description of AIDS (acquired immunodeficiency syndrome) emerged in 1981, marking the beginning of the HIV (Human Immunodeficiency Virus) pandemic. Individuals at elevated risk of HIV acquisition are predominantly men having sex with men (MSM), individuals engaged in sex work, individuals involved in unprotected sex, and injection drug users. These elevated risks stem primarily from transmission-facilitating behaviors (Kteily-Hawa et al., 2022; He et al., 2020). Public health campaigns and social activism have since improved awareness of transmission routes and treatment options. It also led to increased testing rates (Mall et al., 2012).

Early HIV Infection

We often describe early HIV infection as acute HIV infection (AHI). The virus enters the body via the exposure site and spreads along the lymphoid tissue. The end of an early stage of HIV infection is not easily determined, as it depends on the concentration of viral markers and antibodies in the patient's blood (Suanzes et al., 2023). It usually lasts for a few weeks. Patients with acute HIV infection demonstrate markedly higher transmission potential than those with chronic infection, with viral loads in blood and mucosal secretions reaching levels that may increase infectiousness 26-fold. This heightened risk stems principally from the explosive viral replication characteristic of early infection. (Miller et al., 2010). The heightened infectiousness during acute and early-stage HIV infection accounts for a disproportionate share of incident cases, with epidemiological data suggesting approximately 38% of new transmissions in particular cohorts originate from this phase (Powers et al., 2011).

The initial phase of HIV disease demonstrates rapid viral replication and widespread viral dissemination throughout the body, with particular tropism for CD4+ lymphocytes, resulting in their gradual depletion and subsequent immunodeficiency (Weber, 2001). During transmission and early infection, the virus primarily uses the CCR5 co-receptor, later evolving to infect new cell types using CXCR4, which influences disease progression and viral tropism (Swanstrom & Coffin, 2012). During the early phase of HIV infection, high levels of viremia are present, which triggers a strong immune response, often accompanied by acute retroviral syndrome (Daar, 1998).

Clinical Presentation of Early HIV Infection

The clinical presentation of early HIV infection, or acute retroviral syndrome (ARS), typically occurs 2 to 4 weeks (ranging between 4 days and 8 weeks) after the exposure and lasts about 2 to 4 weeks, though symptom duration can vary widely. Common symptoms include fever, chills, night sweats, muscle aches, sore throat, fatigue, swollen lymph nodes, mouth ulcers, headache, joint pain, nausea, diarrhea, rash, and malaise (Swinkels et al., 2025). The syndrome resembles a nonspecific flu-like or mononucleosis-like illness, which makes early diagnosis challenging due to overlap with other common viral infections such as influenza. Fever and rash are among the best clinical predictors of ARS. Oral ulcers and weight loss are more specific symptoms, but occur less frequently (Cowan et al., 2024). Symptoms like diarrhea and nausea, as well as neurological complaints like headaches and dizziness, are also frequently reported during ARS (Crowell et al., 2018).

Common Hematological Abnormalities in Early HIV Infection

1. Anemia

Anemia frequently develops during initial HIV infection and is the most common hematological aberration. Its prevalence rates range widely. In accordance with observational studies, the prevalence among adults infected with HIV ranges from 46.6 to 55.15% (Cao et al., 2022). The anemia observed in early HIV infection is typically normocytic and normochromic, with trends toward iron deficiency indicated by microcytosis, reflecting impaired hematopoiesis and inflammation caused by viral replication and immune activation (Bhardwaj et al., 2020). In acute HIV infection, hemoglobin levels tend to decline early, with studies showing an increase in anemia prevalence from about 25% before infection to over 50% within the first year. Anemia during early HIV infection may results from multifactorial causes such as direct viral suppression of hematopoiesis, immune-mediated destruction, nutritional deficiencies, and inflammation-driven iron dysregulation. Recognition of anemia in early HIV is essential because it may predict rapid disease progression and influence decisions about antiretroviral therapy initiation and supportive care (Mlisana et al., 2008). HIV-associated anemia significantly worsens clinical outcomes, accelerating disease progression while increasing both morbidity and mortality risks. Patients with advanced immunosuppression (low CD4 counts) experience particularly severe impacts on the quality of life (Meidani et al., 2012).

2. Leukopenia and neutropenia

Low white blood cell and neutrophil counts commonly occur in HIV patients, mainly due to the virus suppressing bone marrow function and disrupting normal immune regulation. Leukopenia is a well-documented hematologic abnormality in HIV infection, especially in patients with advanced stages of the disease. Research demonstrates that leukopenia and neutropenia exhibit variable prevalence rates in treatment-naïve patients, typically resolving following successful ART initiation. It is significantly associated with low CD4+ T-cell counts. Three main mechanisms drive these hematologic changes: (1) direct viral suppression of bone marrow function, (2) secondary opportunistic infections, and (3) medication side effects (Tilahun et al., 2022).

Among white blood cell deficiencies, neutrophil depletion occurs most frequently, affecting 5-30% of patients with early symptomatic HIV and rising to 70% in advanced AIDS cases. This HIV-associated neutropenia increases the risk of bacterial infections. It arises from multiple factors, including the direct effects of HIV infection, autoimmune conditions, opportunistic infections, and the medications employed to manage both HIV and its related infections (Damtie et al., 2021).

Leukopenia and neutropenia during early HIV infection serve as critical prognostic markers. These cytopenias contribute significantly to morbidity in HIV-infected individuals by predisposing them to opportunistic and bacterial infections. A decline in the neutrophil count in combination with the failure of adaptive immunity, makes the host highly susceptible to developing fatal infections (Shi et al., 2014)

3. Lymphopenia

Early HIV infection rapidly and significantly depletes CD4+ T cells, causing marked lymphopenia, which plays a crucial role in the progression of immunodeficiency. Extensive CD4+ memory T-cell destruction can be observed quite early during primary HIV infection, almost always without apparent immunodeficiency (Okoye & Picker, 2013). Early decline in CD4+ T-cell counts during early HIV infection serves as a strong predictor of faster disease progression and adverse clinical outcomes (Kaufmann et al., 1999). In early HIV infection, CD4+ T-cell depletion is accompanied by intense immune activation, with CD8+ T cells rapidly expanding and expressing activation markers, which correlate with viral control and disease progression (Streeck & Nixon, 2010). Functional impairments in CD4+ lymphocytes also increase susceptibility to opportunistic infections.

B-cell abnormalities are evident early, including reduced peripheral B-cell counts and a shift toward increased plasmablasts and resting memory B cells. While antiretroviral therapy induces partial B-cell recovery, these abnormalities persist to some degree (Moir et al., 2010). In the acute phase we observe a cytokine storm and elevated proinflammatory cytokines IL-6, IL-10, IFN-γ, and TNF-α. It exacerbates immune dysfunction and lymphocyte apoptosis (Rychert et al., 2010).

Acute HIV-1 infection activates NK cells, which in vitro, kill cells infected with the virus. The early activation of natural killer (NK) cells shows insufficient control of viral replication. This process drives progressive CD4+ T-cell loss and sustains viral reservoirs (Cohen et al., 2011). Early antiretroviral intervention helps contain viral seeding of reservoirs while dampening chronic immune activation, leading to better prognostic outcomes. The depletion of CD4+ lymphocytes and associated immune perturbations fundamentally shape HIV's pathogenic mechanisms during acute infection.

4. Thrombocytopenia

Low platelet counts rank as the second most frequent blood disorder observed during initial HIV infection. It affects around 4% to 40% of patients. In 10% of thrombocytopenic cases, low platelet counts represented the first clinical sign of HIV infection prior to HAART initiation, prompting subsequent HIV diagnosis (Getawa et al., 2021).

Immune thrombocytopenic purpura (ITP) affects up to 30% of people infected with it and can be the first clinical manifestation of HIV infection. HIV-induced autoantibodies mediate premature immune-mediated destruction of platelets. Diagnosis requires the exclusion of pseudothrombocytopenia and other causes, with bone marrow biopsy reserved for refractory cases. The treatment centers on antiretroviral therapy (ART) combined with ITP-specific therapies such as corticosteroids or intravenous immunoglobulins.

Acquired thrombotic thrombocytopenic purpura (TTP) in untreated HIV is caused by platelet microthrombi forming in small blood vessels, which mechanically damage red blood cells, resulting in hemolysis and organ ischemia. Laboratory findings typically show severe thrombocytopenia (often below 30×10^9/L), anemia, and fragmented red cells, although the full clinical pentad of TTP symptoms may not always be present. Treatment consists of starting antiretroviral therapy, corticosteroids, and daily plasma infusions (Opie et al., 2024). Antiretroviral therapy (ART) significantly improves platelet counts and reduces the prevalence of thrombocytopenia in HIV-infected individuals.

Clinical Manifestations and Diagnostic Evaluation

Initial HIV infection typically presents vague, flu-like symptoms - fever, rash, swollen lymph nodes, sore throat, myalgia, and unexplained weight loss. These nonspecific manifestations frequently lead clinicians to suspect common viral infections like influenza or mononucleosis, often delaying HIV detection. Laboratory findings frequently show leukopenia, thrombocytopenia, and elevated liver transaminases. The viral load during acute infection is usually very high, and the CD4 count declines. Diagnosis relies on sensitive assays detecting HIV RNA or p24 antigen before antibody seroconversion. In the early phase of infection, conventional antibody tests may be negative. Because symptoms overlap with other diseases, clinicians should be cautious with patients with recent high-risk exposures and use combination antigen/antibody assays followed by HIV RNA testing when indicated. The characteristics of the included studies are presented in Table 1, and the main findings are summarized in Table 2.

Table 1: characteristics of the included studies

Study (Citation)	Aim	Outcome	Main Findings
Wright (2013)	Examine HIV testing barriers	Significant social stigma impacts early diagnosis	Stigma reduction crucial for improving testing accessibility
Miller et al. (2010)	Assess transmission risk in AHI	26× higher transmission risk during acute infection	38% of new transmissions originate from acute HIV phase
Swanstrom & Coffin (2012)	Characterize viral tropism	CCR5→CXCR4 co-receptor switch	Co-receptor usage evolution impacts disease progression
Swinkels et al. (2024)	Describe ARS symptoms	Fever the most common	Symptoms appear 2-4 weeks post-exposure
Cao et al. (2019)	Determine anemia prevalence	46.6-55.15% in early HIV	Normocytic/normochromic pattern most common
Tilahun et al. (2022)	Evaluate leukopenia prevalence	5-30% in early HIV, up to 70% in AIDS	Associated with low CD4+ counts, improves with ART
Getawa et al. (2021)	Assess thrombocytopenia	4-40% prevalence, ITP in 30%	May be initial HIV manifestation in 10% of cases
Rosenberg et al. (2015)	Compare testing methods	RNA/p24 positive before seroconversion	Combo antigen/antibody assays + RNA testing recommended

Table 2: main findings of the included studies.

Abnormality	Pathophysiology	Clinical Presentation	Diagnostic Approach	Management Considerations
Anemia	- Chronic inflammation - Impaired erythropoiesis - Nutritional deficiencies (iron/B12/folate) - Direct viral bone marrow suppression	- Fatigue, pallor - Reduced exercise tolerance - Worse prognosis in advanced HIV	- CBC (normocytic/normochromic or microcytic) - Iron studies, ferritin, B12/folate levels - Reticulocyte count	- Address underlying cause (e.g., iron supplementation) - Optimize ART - Consider erythropoietin in severe cases
Leukopenia/Neutropenia	- Bone marrow suppression by HIV - Immune-mediated destruction - Opportunistic infections or drug toxicity	- Increased infection risk (bacterial/fungal) - Fever, recurrent infections - Poor wound healing	- Bone marrow biopsy (if persistent/severe) - Rule out Opportunistic Infections/drug effects	- ART initiation - G-CSF for severe neutropenia - Prophylactic antibiotics
Lymphopenia (CD4+ T-cell depletion)	- Direct HIV cytopathic effect - Apoptosis from immune activation - Lymphoid tissue damage	- Increased susceptibility to Opportunistic Infections - Poor vaccine responses - Correlates with disease progression	- CD4+ T-cell count (early decline) - HIV viral load - Lymphocyte subset analysis	- Immediate ART to preserve immunity - Monitor for Opportunistic Infections
Thrombocytopenia	- Immune-mediated destruction (ITP-like) - Impaired megakaryopoiesis - Secondary TTP/HUS in advanced HIV	- Mucosal bleeding - Rarely severe hemorrhage - May precede HIV diagnosis	- CBC (isolated ↓ platelets) - Peripheral smear (schistocytes if TTP) - HIV serology/p24 antigen	- ART (first-line) - IVIG or steroids for ITP - Plasma exchange for TTP

4. CONCLUSIONS

Anemia, thrombocytopenia, neutropenia, leukopenia, and elevated inflammatory markers are significant hematological markers of disease progression in early HIV infection. Research demonstrates that even patients with stable CD4 counts face increased mortality when inflammatory markers such as CRP remain elevated. Our analysis evaluates hematologic manifestations of early HIV infection, assessing their diagnostic utility as early warning signs. We explore associations between specific blood parameters and disease progression while examining the clinical significance of various hematologic alterations. These findings highlight the importance of including HIV testing in clinical evaluation when characteristic hematologic abnormalities are detected.

Author's Contributions

Conceptualization: Agnieszka Radziwonka; Methodology: Agnieszka Radziwonka; Software: Agnieszka Radziwonka ; Check: Agnieszka Radziwonka; Formal analysis: Agnieszka Radziwonka; Investigation: Agnieszka Radziwonka; Resources: Agnieszka

Radziwonka; Data curation: Agnieszka Radziwonka; Writing - rough preparation: Agnieszka Radziwonka; Writing - review and editing: Agnieszka Radziwonka; Visualization: Agnieszka Radziwonka; Supervision: Agnieszka Radziwonka; Project administration: Agnieszka Radziwonka; Receiving funding - no specific funding. All authors have read and agreed with the published version of the manuscript.

Acknowledgments
We thank all the professors for supporting our medical studies and research.

Informed consent
Not applicable.

Ethical approval
Not applicable.

Funding
This study has not received any external funding.

Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data associated with this study are present in the paper.

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