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ASSOCIATION BETWEEN CD4 T-CELL COUNTS AND HEMATOLOGICAL ABNORMALITIES IN HIV-POSITIVE PATIENTS RECEIVING ANTIRETROVIRAL THERAPY

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Abstract

Background: *Hematological abnormalities are common complications in HIV infection. While cytopenias are common even on ART, the prognosis is better.*

Objective: *To assess relationship of CD4+ T-cell count and hematological parameters in HIV infected people on ART.*

Methods: *A cross-sectional study of 100 HIV-positive adults receiving treatment with ART was undertaken. Patients were stratified by CD4 counts: <200, 200–349, 350–499, and ≥500 cells/μL. ANOVA, chi-square and Pearson/Spearman correlation were used to check for hematological parameters.*

Results: *The overall prevalence of hematological abnormality was 65.0%. Primary anemia (60.0 %), leukopenia (12.0 %) and thrombocytopenia (6.0 %) were the main abnormalities. There was a significant step-wise increase in hemoglobin, RBC count, and HCT in the different ascending CD4 categories ($p<0.0001$). Anemia prevalence was 100.0% at CD4<200 versus 0.0% at CD4≥500 (Chi-Square=73.19, $p<0.0001$). Thrombocytopenia occurred*

exclusively in CD4<200 (24.0%, p=0.00025). Strong positive correlation was seen between CD4 and Hb (r=0.931), CD4 and HCT (r=0.902), and CD4 and RBC (r=0.721).

***Conclusions**: There is a strong association between severe CD4 immunosuppression and high prevalence of anemia and low platelet counts. Regular hematological monitoring is still critical for the assessment of the severity of the disease and decision making about treatment.*

INTRODUCTION

Human Immunodeficiency Virus (HIV) infection is an important public health problem worldwide, with an estimated 39 million people infected with HIV globally. The virus infects mainly CD4+ T-lymphocytes, causing a gradual breakdown of the immune system that can result in acquired immune deficiency syndrome (AIDS) if not treated [1]. The disease is prevalent in Sub-Saharan Africa where the vast majority of new HIV infections in the world take place – almost two thirds. HIV pathogenesis is characterized by direct viral killing of CD4+ T lymphocytes, killing of infected cells by apoptosis, and immune-mediated killing of infected cells. As these CD4+ cells also progressively die off, cellular immunity is weakened and susceptible to opportunistic infections and malignancies. In addition to lymphoid system involvement, HIV infection can include involvement of other organs, bone marrow microenvironment impairment being one of the most important aspects of HIV infection. The hematopoietic progenitor cells are affected by the inflammatory milieu created by chronic HIV infection, with cytokines playing pivotal roles in hematopoietic progenitor cells homeostasis and immune reconstitution [2]. Systemic abnormalities are those that occur in other systems of the body and are one of the most commonly seen among them is hematological abnormality. Cytopenias (anemias, leukopenia and thrombocytopenia) are common and can occur as single or as combined abnormalities like pancytopenia throughout the course of the infection. The mechanisms of these hematological features are complex. Bone marrow stromal cells are directly infected by HIV leading to suppression of hematopoiesis; opportunistic infections, peripheral destruction of blood cells by autoimmunity, altered iron metabolism, and drug toxicity all play a role in the development of cytopenias [3].

The approach to HIV in the clinic is not just the cytopathic properties of HIV, but also systemic inflammation, immune dysfunction and opportunistic infections in multiple organ systems. This is especially true in diseases which involve infection of the central nervous system (CNS) such as tuberculous meningitis (TBM) which mainly affects people who are immunocompromised, such as HIV infected people, in which blood-cerebrospinal fluid barrier (BCSFB) integrity is broken [4]. These alterations in physiological barriers, the result of proinflammatory cytokines, including TNF-α and IL-6, illustrate the systemic nature of HIV-related pathology [4]. Likewise, hyperinflammation and immune activation during the course of HIV infection are now known to play a prominent role in the pathogenesis of diverse types of malignancy, including prostate cell malignancies that can develop a self-reinforcing loop of inflammation and epithelial proliferation from the intratumoral microbiota and microbial-androgen crosstalk [5]. These results can help clarify the association of chronic immune activation and the resulting environment that leads to both infectious and non-infectious co-morbidities. Therefore, the search for accessible biomarkers is important for monitoring disease progression. For example, there are different CSF biochemical parameters that differ between TBM patients of different genders and require gender-specific diagnosis; this highlights the necessity of context-appropriate and precise clinical parameters [6]. The paradigm of systemic inflammation and immune dysregulation is essential for understanding the persistence of certain hematological abnormalities, including anemia, leukopenia and thrombocytopenia, in HIV-negative individuals even those who are on successful antiretroviral therapy (ART) [7].

A most common cytopenia is anemia, which is caused by chronic inflammation and increased inflammatory cytokines including TNF-α that depresses levels of EPO and affects the function of erythroid precursor cells. The anemic condition is worsened by coinfections with TB and nutrition deficiencies (parvovirus B19). Immune-mediated destruction of platelets and diminished megakaryocyte function likely underlie the development of thrombocytopenia, with HIV being able to infect

megakaryocytes directly. Leukopenia, especially neutropenia, is due to direct viral suppression of granulocyte precursor cells or immune-mediated destruction of circulating leukocytes [8]. Highly active antiretroviral therapy (HAART), introduced in the late 1990s, has revolutionized the way HIV is managed, and has resulted in a dramatic decrease in viral load, immune recovery and increased survival rates. ART slows down the growth of HIV and allows CD4+T-cells to start rebuilding and the immune system to recover. HIV-1 replication can be suppressed by ART and studies have shown that this reduction of viral replication leads to a restoration of hematopoietic stem cell activity, probably because of the suppression of factors that inhibit normal hematopoiesis [9]. But even with good viral suppression, cytopenias continue in some patients, especially those who do not have optimal viral control or who are most severely immunosuppressed. Even if the virus is suppressed, it may have long-term impact on the hematopoiesis due to continued release of pro-inflammatory cytokines. Further, certain ARTs (especially the older nucleoside reverse transcriptase inhibitors like zidovudine) have been linked to myelosuppression. Persistent cytopenias indicate the need for monitoring of everything hematological, besides CD4 counts, to measure the response of the ART and to manage the patient clinically [10]. The link between HIV infection and hematological abnormalities is now well recognized, but there is still a need for local clinical data that link quantitative CD4 strata to continuous hematological markers. Previous investigations have shown that thrombocytopenia is directly related to low numbers of CD4+ cells and that cytopenias occur more commonly as a result of more severe immunosuppression [11]. But the correlation of CD4 recovery with the resolution of cytopenias in the various hematopoietic lineages needs to be studied further. It is still unknown whether there is a symmetrical recovery of erythroid, myeloid, and megakaryocytic cells along with CD4 recovery and if there are any particular cytopenias that can be used as surrogate markers of immune restoration. This study was therefore aimed at determining the prevalence of anemia, leukopenia and thrombocytopenia across the categories of CD4 and at testing the direct association between CD4 count and hematological indices, with the hypothesis that low CD4 T-cell count is directly correlated with low hemoglobin, hematocrit, red cell and platelet count and with high prevalence of cytopenia [12].

Methodology

1. Study Design and Participant Selection

The study was a cross-sectional analysis (N = 100) of 100 adult patients with HIV currently participating in a HIV care and treatment program (HCT) specifically designed for adults. 100% of the participants were actively on combination Antiretroviral Therapy (ART).

Inclusion Criteria: Confirmed HIV-1/2 diagnosis, age 18 years or older, ongoing ART for 1 month or longer, and availability of complete concurrent CD4 T-cell and hematological profile data.

Exclusion Criteria: Patients with active severe acute sepsis, known active non-HIV malignancies undergoing chemotherapy/radiotherapy, acute overt hemorrhage, recent blood transfusion within 90 days prior to sampling, pregnancy, or known underlying primary hematological disorders (e.g., hemoglobinopathies, idiopathic thrombocytopenic purpura).

2. Laboratory Data Collection and Diagnostic Measurements

Peripheral venous blood samples were collected into K2-EDTA vacuum tubes under standard aseptic venipuncture techniques.

Immunological Assessment: CD4+ T-lymphocyte counts (cells/μL) were quantified using automated flow cytometry (BD FACSCalibur / FACSCount system).

Hematological Assessment: Complete Blood Count (CBC) was performed using an automated 5-part differential hematology analyzer. Parameters quantified included:

- **Erythroid Lineage:** Hemoglobin (Hb, g/dL), Red Blood Cell Count (RBC, × 10^12/L), Hematocrit (HCT, %), Mean Corpuscular Volume (MCV, fL).
- **Myeloid Lineage:** Total White Blood Cell count (WBC, × 10^3/μL), Relative Neutrophil Count (%), Relative Lymphocyte Count (%).
- **Megakaryocytic Lineage:** Platelet Count (× 10^3/μL).

3. Diagnostic Definitions and Categorization Standards

Patients were stratified into four distinct CD4 T-cell immunological categories according to modified WHO/CDC classification guidelines:

CD4 Category	Immunological Status
CD4 < 200 cells/μL	Severe Immunosuppression
CD4 200–349 cells/μL	Moderate Immunosuppression
CD4 350–499 cells/μL	Mild Immunosuppression
CD4 ≥ 500 cells/μL	Normal / Restored Immune Function

Hematological abnormalities were defined according to standard laboratory clinical threshold criteria:

- **Anemia:** Hemoglobin < 12.0 g/dL
- **Leukopenia:** Total WBC count < 4.0 × 10^3 cells/μL
- **Thrombocytopenia:** Platelet count < 150 × 10^3 cells/μL

4. Statistical Analysis Plan

Statistical processing and data analysis were conducted using Python (v3.11) with pandas, SciPy. stats, matplotlib, and seaborn statistical modules.

Descriptive Statistics: Continuous variables were checked for normality (Shapiro-Wilk test). Parametric variables were presented as Mean ± Standard Deviation (SD), and categorical variables were expressed as frequencies and percentages (n, %).

Inferential Comparative Statistics: Continuous hematological variables across the four CD4 categories were evaluated using one-way Analysis of Variance (ANOVA). Categorical abnormality rates across CD4 categories were evaluated using Pearson's Chi-Square test of independence.

Correlation and Regression: Association between continuous CD4 counts and hematological indices was quantified using Pearson product-moment correlation coefficient (r) and Spearman rank correlation (ρ). Simple linear regression models were fitted to visualize linear trajectories.

Significance Level: A two-tailed p-value less than 0.05 was considered statistically significant for all analyses.

Results

1. Baseline Demographic and Clinical Characteristics

A total of 100 HIV infected patients on ART were included in the study comprising 51(51.0%) males and 49(49.0%) females. The overall cohort had a mean age of 41.36 ± 11.71 years (range: 20–60 years) and a mean ART duration of 43.04 ± 22.31 months (range: 1–84 months). The overall mean CD4+ T-cell count was 338.46 ± 176.08 cells/μL.

Immunological classification of the 100 patients showed that 25 (25.0%) were in the severe CD4 subgroup (< 200 cells/μL), 29 (29.0%) were in moderate subgroup (200-349 cells/μL), 32 (32.0%) in mild subgroup (350-499 cells/μL), and 14 (14.0%) in non-immunosuppressed group (≥ 500 cells/μL). The mean age or the mean duration of ART were similar and did not differ significantly among the four CD4 groups (p = 0.611 and 0.349 respectively). Hundred percent demographic and laboratory baseline data have been provided (see Table 1).

Table 1: Baseline Demographic, Clinical, and Laboratory Parameters Stratified by CD4 T-Cell Categories (N=100)

Parameter	Overall Cohort (N=100)	CD4 < 200 (n=25)	CD4 200–349 (n=29)	CD4 350–499 (n=32)	CD4 ≥ 500 (n=14)	F-Statistic	p-value
Male / Female (n, %)	51 / 49	15 (60%) / 10 (40%)	18 (62.1%) / 11 (37.9%)	12 (37.5%) / 20 (62.5%)	6 (42.9%) / 8 (57.1%)	-	-

Age (years)	41.36 ± 11.71	41.80 ± 11.05	38.97 ± 12.01	42.91 ± 11.19	42.00 ± 13.78	0.61	0.6110
ART Duration (months)	43.04 ± 22.31	46.88 ± 25.77	36.90 ± 20.16	44.19 ± 19.42	46.29 ± 25.73	1.11	0.3486
CD4 Count (cells/µL)	338.46 ± 176.08	135.80 ± 38.65	274.28 ± 49.40	414.41 ± 42.26	659.71 ± 101.55	382.41	< 0.0001
Hemoglobin (g/dL)	11.73 ± 1.47	10.21 ± 0.66	11.10 ± 0.52	12.41 ± 0.60	14.21 ± 1.02	127.52	< 0.0001
RBC Count (× 10^12/L)	4.38 ± 0.19	4.21 ± 0.13	4.32 ± 0.12	4.45 ± 0.14	4.61 ± 0.15	29.93	< 0.0001
Hematocrit (%)	35.29 ± 4.68	30.76 ± 2.29	33.19 ± 2.14	37.25 ± 2.21	43.27 ± 2.91	103.02	< 0.0001
MCV (fL)	88.66 ± 5.49	88.54 ± 5.02	87.34 ± 5.65	89.94 ± 5.25	88.65 ± 6.40	1.14	0.3360
WBC (× 10^3/µL)	5.81 ± 1.52	5.64 ± 1.62	6.08 ± 1.66	5.73 ± 1.38	5.75 ± 1.45	0.44	0.7272
Platelets (× 10^3/µL)	241.46 ± 73.63	192.12 ± 60.32	262.31 ± 72.03	257.00 ± 67.56	250.86 ± 78.53	5.81	0.0011

2. Distribution of Hematological Parameters Across CD4 Categories

However, significant step-wise changes in the erythroid parameters were noted with increasing CD4 cell counts ($p < 0.0001$). Mean hemoglobin levels rose systematically across ascending CD4 categories: 10.21 ± 0.66 g/dL in the < 200 group, 11.10 ± 0.52 g/dL in the 200–349 group, 12.41 ± 0.60 g/dL in the 350–499 group, and reached 14.21 ± 1.02 g/dL in the ≥ 500 group ($F = 127.52$, $p < 0.0001$). Similarly, red blood cell counts increased from 4.21 ± 0.13 × 10^12/L to 4.61 ± 0.15 × 10^12/L ($F = 29.93$, $p < 0.0001$), and hematocrit increased from 30.76 ± 2.29% to 43.27 ± 2.91% ($F = 103.02$, $p < 0.0001$).

Platelet counts exhibited significant variance across CD4 groups ($F = 5.81$, $p = 0.0011$), with lower mean counts in the severe CD4 subgroup (192.12 ± 60.32 × 10^3/µL) compared to the higher CD4 categories (262.31 ± 72.03 × 10^3/µL in the 200–349 group). There was no significant difference in the number of WBCs and MCV between different CD4 levels ($p = 0.727$ and 0.336, respectively).

3. Prevalence of Hematological Abnormalities

For all patients, there were at least one hematological abnormality in 65/100 patients (65.0%). The most common abnormalities were anemia (60.0%, n = 60), leukopenia (12.0%, n = 12) and thrombocytopenia (6.0%, n = 6).

There was significant difference in prevalence of anemia by CD4 strata (Chi-Square = 73.19, $p < 0.0001$). Anemia was present in 100.0% (25/25) of patients with CD4 < 200 cells/µL and 96.6% (28/29) of those with CD4 200–349 cells/µL, falling to 21.9% (7/32) in the 350–499 CD4 group, and 0.0% (0/14) in patients with CD4 ≥ 500 cells/µL.

All patients with thrombocytopenia had CD4 counts below 200 cells/µL (6/25, 24.0%) and this was found to be significantly associated with severe immunosuppression (Chi-Square = 19.15, $p = 0.00025$). No significant difference was found between leukopenia in different CD4 categories (Chi-Square = 0.37, $p = 0.9471$). The comparative prevalence and chi-square test scores are shown in detail in Table 2.

Table 2: Prevalence of Cytopenias and Correlation Coefficients with CD4 Count

Outcome / Abnormality	Overall (N=100)	CD4 < 200 (n=25)	CD4 200–349 (n=29)	CD4 350–499 (n=32)	CD4 ≥ 500 (n=14)	Chi-Square Statistic	Prevalence p-value	Pearson r with CD4	Pearson p-value	Spearman ρ with CD4

Anemia (Hb < 12.0 g/dL)	60 (60.0 %)	25 (100.0 %)	28 (96.6 %)	7 (21.9 %)	0 (0.0 %)	73.19	< 0.0001	0.931	< 0.0001	0.925
Leukopenia (WBC < 4.0 × 10^3)	12 (12.0 %)	3 (12.0 %)	4 (13.8 %)	3 (9.4 %)	2 (14.3 %)	0.37	0.9471	-0.019	0.8535	0.006
Thrombocytopenia (Plt < 150 × 10^3)	6 (6.0 %)	6 (24.0 %)	0 (0.0 %)	0 (0.0 %)	0 (0.0 %)	19.15	0.00025	0.204	0.0422	0.235
Any Cytopenic Abnormality	65 (65.0 %)	25 (100.0 %)	28 (96.6 %)	10 (31.2 %)	2 (14.3 %)	58.00	< 0.0001	-	-	-
RBC Count	-	-	-	-	-	-	-	0.721	< 0.0001	0.685
Hematocrit (HCT)	-	-	-	-	-	-	-	0.902	< 0.0001	0.886

4. Bivariate Correlation and Visualization Analysis

Bivariate correlation analysis revealed strong linear relationships between CD4 counts and red cell parameters. CD4 cell count showed a positive correlation with hemoglobin level ($r = 0.931$, $p < 0.0001$; Spearman $\rho = 0.925$), hematocrit ($r = 0.902$, $p < 0.0001$), and RBC count ($r = 0.721$, $p < 0.0001$). Platelet count demonstrated a weak positive correlation with CD4 count ($r = 0.204$, $p = 0.0422$). Neither total WBC count ($r = -0.019$, $p = 0.8535$) nor ART duration ($r = 0.033$, $p = 0.7447$) correlated significantly with CD4 levels.

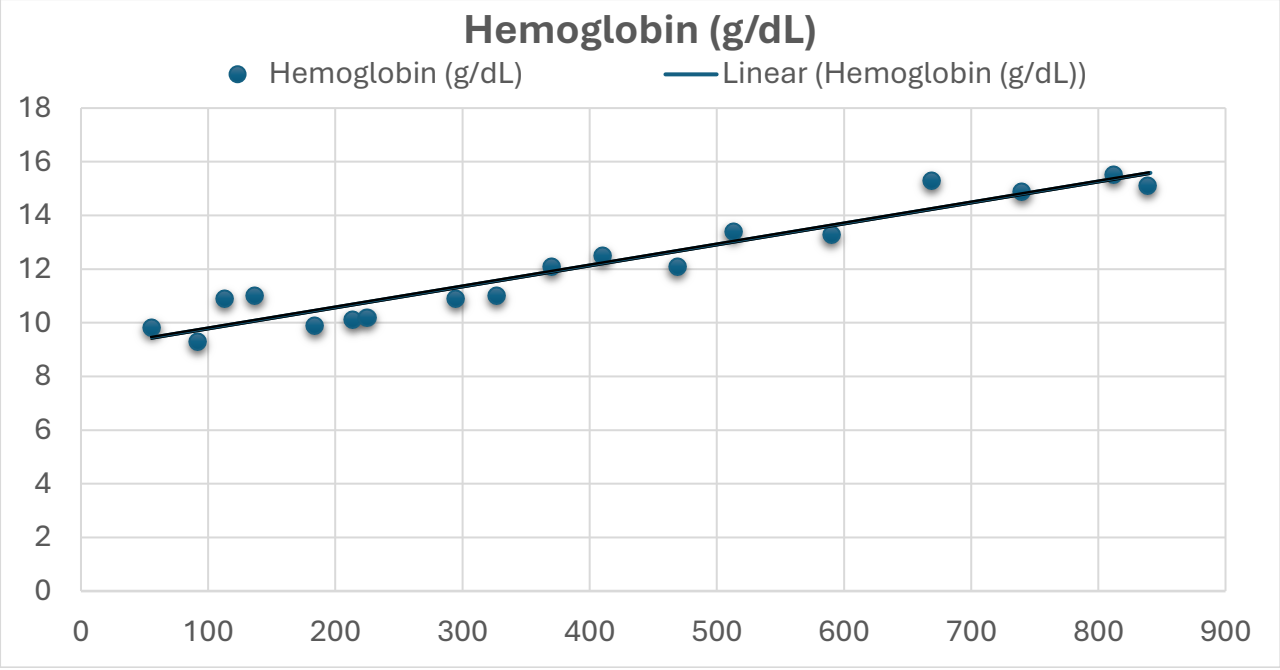


Figure 1: Scatter Plot Showing Correlation Between CD4 T-Cell Count and Hemoglobin Level

Scatter plot with CD4 count on x-axis and Hemoglobin on y-axis, showing positive linear relationship ($r = 0.931$), with anemia threshold line at 12 g/dL and severe immunosuppression threshold line at 200 cells/μL

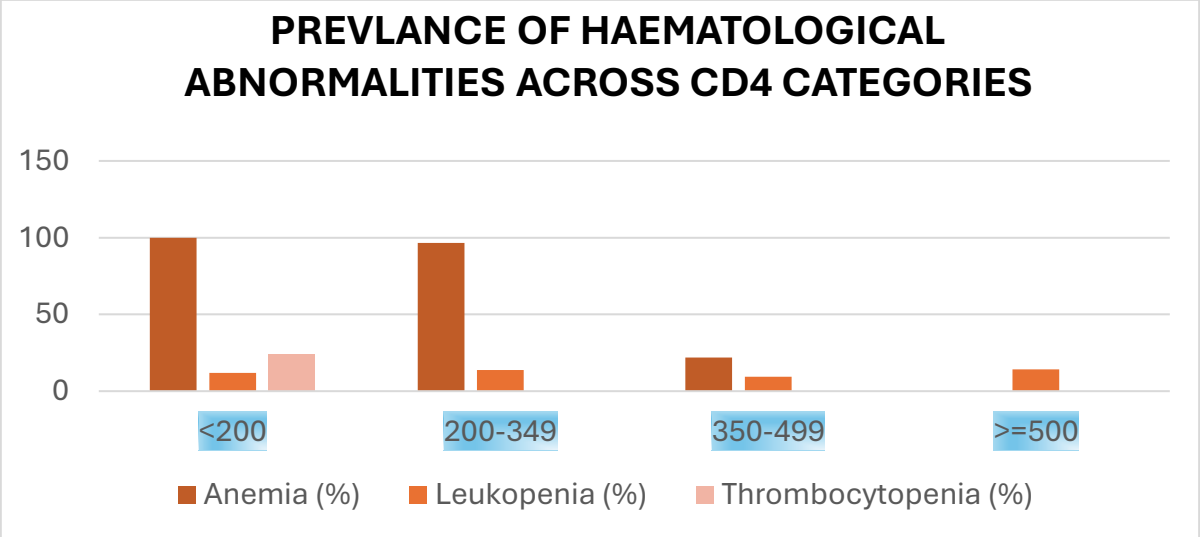


Figure 2: Prevalence of Hematological Abnormalities Across CD4 T-Cell Strata

Bar chart showing comparative prevalence of anemia, leukopenia, and thrombocytopenia across CD4 categories: CD4 < 200, 200–349, 350–499, and ≥ 500 cells/μL

Discussion

This study demonstrates that hematological abnormalities remain highly prevalent (65%) among ART-treated HIV-positive patients, with anemia dominating as the primary cytopenia (60%). The strong direct correlation between CD4 count and red cell parameters (Hb r = 0.931, HCT r = 0.902) confirms that immune restoration, as reflected by rising CD4 counts, is closely linked to recovery of erythroid lineage function. These findings align with previous research demonstrating that cytopenias, particularly anemia, are the most frequently observed hematological abnormalities in HIV patients, with multiple underlying mechanisms including chronic inflammation, direct viral effects, and drug toxicity [13]. The high prevalence of anemia in CD4<200 (100 %), and the absence of anemia in CD4 ≥500 (0 %) highlights the effect of severe immunosuppression on the erythropoietic function. This finding is in line with the concept that direct HIV infection of bone marrow stromal cells lowers the activity of erythropoiesis at low CD4 counts. In severe immunosuppression, in addition, insufficient production of erythropoietin (EPO) for the severity of the anemia is a factor [14]. Iron dysregulation leading to anemia of chronic disease is due to chronic inflammation in which there is increased IL-6 and TNF-alpha and hepcidin is upregulated. Complete normalization of hemoglobin levels at CD4 ≥ 500 cells/μL suggests that recovery of the production of red blood cells is in parallel with immune reconstitution, but the presence of persistent cytopenias at normalized CD4 counts underline that other factors remain contributing [15]. Thrombocytopenia was only present in those with CD4 levels <200 cells/μL (24% prevalence) reflecting a strong association between severe immunosuppression and abnormalities of platelets. This finding supports more recent research that found low CD4+ T-cell count to be a strong indicator of thrombocytopenia in individuals with HIV [16].

The mechanisms may include direct infection of megakaryocytes leading to a decrease in platelet production and cross-reactive platelet destruction through autoimmune mechanisms. The complete resolution of thrombocytopenia in patients with CD4 count ≥ 500cell/μL is indicative of the reversibility of this condition and this has been seen in different studies in which the thrombocytopenia reversed after HAART in 100 % of patients [17]. The results of comparing the counts of myeloid-lineage cells (WBC and neutrophils) and CD4 level did not show any significant correlation (r = -0.019), indicating perhaps a different recovery path for the myeloid-lineage cells, as compared with the erythroid and megakaryocytic. This could also be due to the effect of some of the ART medications on the bone marrow lines; older nucleoside reverse transcriptase inhibitors (NRTIs), like zidovudine, are known to cause myelosuppression [18]. Drug-related bone marrow suppression, even in the context of viral suppression, may explain why leukopenia persists regardless of immunological status. Additionally, neutropenia in

HIV can result from direct viral suppression of granulocyte precursors or immune-mediated destruction of circulating leukocytes, factors that may not correlate directly with CD4 levels. The consistent leukopenia prevalence (approximately 12%) across all CD4 categories further supports the hypothesis that myeloid suppression may be driven primarily by drug effects rather than immune status alone [19].
Hemoglobin and CD4 count were highly correlated, supporting the use of hemoglobin as an easily available alternative laboratory parameter to monitor immune restoration, especially in resource-limited environments where CD4 count is not readily available. The study results highlight the need for prompt initiation of ART early, before CD4 counts fall below 200 counts/μL, as this could result in severe hematological morbidity [20]. Even if the viral load is undetectable, viral effects on erythrocytes may occur depending on the sex and the ART type, so that hematological follow-up should be maintained in order to provide timely follow-up and individualization of the intervention. A significant number of patients with normal CD4 count (> or equal to 500 cells/μL) had cytopenic abnormalities (14.3%), indicating that factors other than immune function such as drug toxicity, nutrients deficiency and co-infections continue to play a great role in the full care of HIV-positive patients [21].

Strengths and Limitations

The advantages of this research are strict statistical testing, complete full CBC line analyses, the same laboratory-based measurements and the clear stratification in internationally accepted immunological categories. Using both Pearson and Spearman correlation coefficients meant that there was good quantification of the relationships between CD4 counts and hematological parameters. The cross-sectional study design, however, does not allow causal conclusions about the direction of the association between CD4 count and hematological parameters. Detailed baseline viral load data are missing, as are breakdowns of individual drug regimens, which would have enabled more nuanced analysis of the impact of the different ARTs on bone marrow function. Further, the small sample size (N=100) and single center study design constrain the applicability of the findings to other populations. Another limitation is the lack of nutrition status evaluation, iron studies and vitamin B12 and folate levels which could have an independent effect on hematological parameters. Incorporation of longitudinal design in future research would be useful to determine the course of recovery from cytopenia after particular changes in ART and viral load control.

Conclusion

In the ART-treated HIV-positive people, hematological abnormalities, especially anemia and thrombocytopenia, are strongly associated with the low level of CD4+ T cells. The hemoglobin is completely normalized, and the thrombocytopenia is resolved, when the CD4 level rises to over 500 cells/μL in this population. The results are consistent with the idea that normal hematopoiesis is suppressed by factors that are directly or indirectly involved in controlling HIV-1 replication and that these factors can be suppressed by maintaining the virus in its latent state using ART. Hemoglobin and hematocrit are useful markers in addition to the CD4 count to track patients' clinical and hematological recovery. The good correlation between CD4 count and red cell parameters implies that Hemoglobin Monitoring could be a readily available surrogate marker for immune restoration in resource-poor environments.
Routine hematological monitoring should be part of routine ART monitoring algorithms to allow early detection and management of cytopenias. Clinicians should be aware of possible hematological complications in individuals with CD4 cell counts of less than 200 cells/μL and early initiation of ART should be considered to prevent severe hematological morbidity. Future longitudinal studies should focus on the pattern of recovery of cytopenia, after specific changes in ART therapy, and viral load suppression as well as the effects of specific antiretroviral drugs on bone marrow function per lineages and the differential pattern of recovery. Furthermore, with the use of nutritional evaluations, iron indices and inflammatory markers, a more multifaceted picture of the multifactorial genesis of cytopenias in HIV infected patients on ART could be obtained.
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REFERENCES

[1] P. Sarma, J. N. Saikia, A. Hussain, U. Borah, and J. S. Roy, "Understanding HIV: A Review of Pathogenesis and Therapeutics," *J. Adv. Biol. Biotechnol.*, vol. 28, no. 2, pp. 520–525, Feb. 2025, doi: 10.9734/jabb/2025/v28i22012.

[2] F. Gomez-Rivera, "HIV Infection Dynamics: From Underlying the Establishment and Maintenance to the Molecular Mechanisms of Latency," University of Michigan, 2025. doi: 10.7302/25778.

[3] E. I. Obeagu, O. G. Goryavheva, and M. A. Zubarev, "Cytopenias in context: ethnoracial hematological trends among HIV-infected Africans and Russians: a review," *Ann. Med. Surg.*, vol. 88, no. 1, pp. 610–620, Jan. 2026, doi: 10.1097/MS9.0000000000004520.

[4] Muhammad Ehsan *et al.*, "Disruption of the Blood–CSF Barrier in Tuberculous and Non-Tuberculous Meningitis: Mechanisms, Diagnosis, and Clinical Impact," *Wah Acad. J. Health Nutr.*, vol. 1, no. 2, pp. 41–46, Jun. 2025, doi: 10.63954/67v24t98.

[5] Muhammad Faizan, Ahram Hussain, Ahmad Ashraf, Mutahar A Tunio, and Abdul Ahad Mehboob, "The Intratumoral Microbiota in Prostate Cancer: Mechanisms, Microbial–Androgen Crosstalk, and Urinary Exfoliative Signatures for Liquid Biopsy Development," *Wah Acad. J. Health Nutr.*, vol. 1, no. 4, pp. 85–94, Dec. 2025, doi: 10.63954/373g0373.

[6] Ahram Hussain, Tasra Bibi, Syed Wajeeh Ul Hassan, Muhammad Faizan, Faiqa Shakeel, and Abdul Ahad Mehboob, "Tuberculosis Meningitis: An Exploration of Cerebrospinal Fluid Biochemical Markers and Their Correlation with Gender and Age," *Wah Acad. J. Health Nutr.*, vol. 1, no. 3, pp. 51–57, Sep. 2025, doi: 10.63954/7smh9e20.

[7] X. Shen *et al.*, "Characterizing pancytopenia in a large Asian cohort of people living with HIV: A 23-year nested case-control study," *J. Infect. Public Health*, vol. 18, no. 8, p. 102832, Aug. 2025, doi: 10.1016/j.jiph.2025.102832.

[8] P. Li *et al.*, "Associated factors and prognostic implications of neutropenia in individuals with HIV/AIDS," *Virol. J.*, vol. 22, no. 1, p. 6, Jan. 2025, doi: 10.1186/s12985-025-02624-x.

[9] A. E. Myhre *et al.*, "Long-term HIV-1 remission achieved through allogeneic haematopoietic stem cell transplant from a CCR5Δ32/Δ32 sibling donor," *Nat. Microbiol.*, vol. 11, no. 5, pp. 1374–1386, Apr. 2026, doi: 10.1038/s41564-026-02304-8.

[10] X. Shen *et al.*, "Characterizing pancytopenia in a large Asian cohort of people living with HIV: A 23-year nested case-control study," *J. Infect. Public Health*, vol. 18, no. 8, p. 102832, Aug. 2025, doi: 10.1016/j.jiph.2025.102832.

[11] Abdul Rehman, Afia Sarwar, Imtiaz Bashir, Sohail Akram, Amna Ali, and Muhammad Shoaib, "Hematological Derangement in Adult Male HIV/AIDS Patients and Their Relationship with the CD4+ Counts: A Cross-Sectional Study," *J. Health Wellness Community Res.*, p. e195, May 2025, doi: 10.61919/tap67103.

[12] A. Chauhan and A. Banerji, "Hematological Profile in HIV-Positive Patients: A Cross-Sectional Analysis from a Tertiary Care Centre," *Acta Medica Int.*, vol. 12, no. 2, pp. 1–4, Aug. 2025, doi: 10.21276/amit.2025.v12.i2.1.

[13] P. Moreno Fonseca *et al.*, "Erythrovirus-B19-Induced Pure Red Cell Aplasia Revealing Untreated HIV Infection," *Am. J. Case Rep.*, vol. 27, May 2026, doi: 10.12659/AJCR.952075.

[14] C. Anyelwisye *et al.*, "Anemia in HIV-Infected Children on ART in Uganda: High Risk Associations with Malaria, Opportunistic Infections, and Poor Adherence," *HIVAIDS - Res. Palliat. Care*, vol. Volume 18, pp. 1–10, Apr. 2026, doi: 10.2147/HIV.S609192.

[15] P. Moreno Fonseca *et al.*, "Erythrovirus-B19-Induced Pure Red Cell Aplasia Revealing Untreated HIV Infection," *Am. J. Case Rep.*, vol. 27, May 2026, doi: 10.12659/AJCR.952075.

[16] Wiratama Hadi Tjuanda and Anak Agung Ayu Yuli Gayatri, "Unmasking Advanced HIV Infection: A Case of Refractory Thrombocytopenia Misdiagnosed as Immune Thrombocytopenic Purpura," *Arch. Med. Case Rep.*, vol. 6, no. 3, pp. 1437–1447, Jul. 2025, doi: 10.37275/amcr.v6i3.777.

[17] Z. Tang *et al.*, "Analysis of Manifestations and Associated Factors of HIV-1 Associated Thrombocytopenia in a General Teaching Hospital in Western China," *Infect. Drug Resist.*, vol. Volume 18, pp. 1913–1921, Apr. 2025, doi: 10.2147/IDR.S517427.

[18] C. Anyelwisye *et al.*, "Anemia in HIV-Infected Children on ART in Uganda: High Risk Associations with Malaria, Opportunistic Infections, and Poor Adherence," *HIVAIDS - Res. Palliat. Care*, vol. Volume 18, pp. 1–10, Apr. 2026, doi: 10.2147/HIV.S609192.

[19] H. K.-A. Ramadan *et al.*, "First report on leucocytes cell population data as an applicable test for clinical stratification in people living with HIV," *Virus Res.*, vol. 367, p. 199716, May 2026, doi: 10.1016/j.virusres.2026.199716.

[20] T. Mou *et al.*, "Clinical events associated with poor CD4+ T-cell recovery in people living with HIV following ART: A systematic review and meta-analysis," *J. Infect.*, vol. 90, no. 2, p. 106414, Feb. 2025, doi: 10.1016/j.jinf.2025.106414.

[21] X. Shen *et al.*, "Characterizing pancytopenia in a large Asian cohort of people living with HIV: A 23-year nested case-control study," *J. Infect. Public Health*, vol. 18, no. 8, p. 102832, Aug. 2025, doi: 10.1016/j.jiph.2025.102832.