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Investigating Reticulocyte Count, Serum Ferritin, and Transferrin in HIV Infected Patients in Owerri, Nigeria

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Abstract

Human immunodeficiency virus (HIV) infection remains a major global health challenge, and hematological abnormalities especially anemia are among its most common complications. Anemia in HIV infected individuals has been associated with disease progression, decreased quality of life, and increased mortality. Iron metabolism disturbances and impaired erythropoiesis are frequently implicated, yet data from many African settings remain sparse. This study evaluated reticulocyte count, serum ferritin, and transferrin levels in HIV infected adults attending Imo Specialist Hospital, Owerri, Nigeria, compared with age and sex matched HIV negative controls. In a comparative cross-sectional design, 30 HIV-positive adults and 30 controls were recruited. Reticulocyte counts were determined by standard microscopy after new methylene blue staining; serum ferritin and transferrin were measured using immunoassays. HIV-infected patients exhibited significantly lower mean reticulocyte counts (0.22)% compared to controls (0.850%,($p < 0.0001$) and transferrin levels (164.50 ± 84.04) mg/dL compared to controls (284.77 ± 89.01) mg/dL, ($p < 0.0001$), and markedly higher serum ferritin (432.70 ± 240.07) ng/mL compared to controls (65.67 ± 49.60) ng/mL, ($p < 0.0001$) compared to the control group. There were no significant differences by sex or across age strata. Correlation analysis did not reveal significant associations between reticulocyte count and either ferritin or transferrin, suggesting that anemia in this context may be multifactorial and not solely due to iron deficiency. The findings support the concept of anemia of chronic disease (ACD) in HIV infection and underscore the need for comprehensive iron status assessment in HIV care.

Keywords

HIV; anemia; reticulocyte count; ferritin; transferrin; Nigeria

1.0 Introduction

Globally, the burden of Human Immunodeficiency Virus (HIV) infection remains substantial, with millions living with the disease, particularly in sub–Saharan Africa. As survival improves due to wider access to antiretroviral therapy (ART),

long-term complications including hematological abnormalities have become increasingly important determinants of morbidity and quality of life. Among these, anemia is the most common hematologic disorder in people living with HIV (PLWHIV) and has been consistently associated with disease progression, opportunistic infections, and increased mortality.4,5

Anemia in HIV is typically multifactorial. Mechanisms include chronic inflammation, opportunistic infections, nutritional deficiencies (including iron deficiency), bone marrow suppression, and direct or indirect effects of HIV on hematopoiesis.3,6,7 Iron homeostasis is often disrupted in HIV infection; elevated serum ferritin and reduced transferrin levels have been reported in several studies, reflecting altered iron storage and transport rather than true iron deficiency.8,9

Ferritin serves as an iron storage protein and also behaves as an acute-phase reactant, rising in response to inflammation. Meanwhile, Transferrin the primary iron transport protein in plasma tends to decrease in chronic inflammatory states due to reduced synthesis and altered iron utilization.[10] Thus, elevated ferritin combined with reduced transferrin may reflect a state of iron sequestration, consistent with anemia of chronic disease (ACD), rather than simple iron deficiency.

The Reticulocyte count provides insight into erythropoietic activity in the bone marrow. Low reticulocyte counts despite anemia suggest suppressed erythropoiesis, which may occur in chronic inflammatory conditions such as HIV, where cytokine-mediated marrow suppression and altered iron kinetics limit red blood cell production.[11]

Although some studies have examined iron indices and anemia in HIV, data remain limited in many African regions, including Nigeria. In addition, many prior works focus on hemoglobin concentration alone, without detailed assessment of iron transport/storage proteins or bone marrow response. A comprehensive evaluation of reticulocyte count, ferritin, and transferrin could improve characterization of anemia type (iron deficiency vs ACD vs mixed) and inform management decisions.

Therefore, this study aimed to measure reticulocyte count, serum ferritin, and transferrin levels in HIV-infected adults in Owerri, Nigeria, comparing them with HIV-negative controls, and to assess the relationship among these parameters.

Anemia is widely recognized as the most common hematologic abnormality in people living with HIV. A recent systematic review and meta-analysis including 110,113 PLWHIV found a pooled prevalence of anemia of 46.6% (95% CI: 41.9–51.4%) among adults (≥15 years) living with HIV.[5] Notably, anemia prevalence remains high despite widespread ART, suggesting that non–treatment-related factors play significant roles. Additional data indicate that severity of anemia (mild, moderate, or severe) persists in a substantial fraction of patients.[5]

In East Africa, meta-analytic data estimated the pooled prevalence of anemia among HIV infected individuals at around 25.3% (95% CI: 20.7–30.0%), though with variability depending on ART status and study population.[12] This variation underscores the influence of demographic, geographic, and clinical factors in anemia prevalence among PLWHIV.

Anemia in HIV may result from diverse mechanisms: nutritional iron deficiency, chronic inflammation, opportunistic infections, ART toxicity, and bone marrow suppression.7,8 In many cases, iron deficiency appears only partially responsible; a substantial proportion of cases may be attributable to iron redistribution (sequestration) secondary to inflammation.[1]

Iron dysregulation in HIV has been documented in several settings. In a cohort from Indonesia, elevated serum ferritin and low soluble transferrin receptor (sTfR) suggested iron redistribution and decreased erythropoietic activity rather than iron deficiency per se.[14] Similarly, older studies showed high prevalence of elevated serum and red-cell ferritin in HIV-infected patients, increasing with disease progression and decreasing CD4 counts.[16]

In a systematic review of iron status among PLWHIV, high serum ferritin was frequently associated with adverse clinical outcomes including increased risk of mortality and tuberculosis regardless of anemia type.[8] However, the review cautioned that ferritin's role as an acute-phase reactant complicates interpretation, especially in the context of chronic inflammation and immune activation.

Moreover, dysregulation of key iron metabolism modulators including disrupted transferrin levels, altered hepcidin regulation, and impaired iron transport has been described even among virologically suppressed HIV-infected individuals, highlighting persistent disturbances in iron homeostasis despite ART.[15] Thus, a combined assessment of ferritin, transferrin, erythropoietic activity (via reticulocyte count), and if possible, soluble transferrin receptor or hepcidin, is needed for accurate characterization of anemia in HIV.

Reticulocyte count is a useful surrogate of bone marrow erythropoietic output. In HIV infection, chronic inflammation, cytokine-mediated suppression, and direct effects on marrow progenitors often result in reduced erythropoiesis, manifesting as low reticulocyte counts despite anemia.[7] [14] In the Indonesian cohort mentioned above, anemia was associated with elevated ferritin but low sTfR and low reticulocyte production, supporting a pattern of iron redistribution and marrow suppression rather than iron deficiency.[14]However, few studies particularly in sub Saharan Africa have simultaneously assessed reticulocyte counts, ferritin, and transferrin (or transferrin-related parameters), limiting understanding of anemia etiology in many settings. This gap is more pronounced in Nigeria, where regional data remain scarce.

Given the high burden of HIV in Nigeria, the frequency of hematologic complications, and the limited data on detailed iron metabolism parameters, there is a need for region specific studies examining anemia etiology. By combining measurements of reticulocyte count, serum ferritin, and transferrin in HIV-infected adults and comparing with matched controls, this study aims to clarify the underlying mechanisms of anemia in this population distinguishing between iron deficiency, iron redistribution, and marrow suppression. Such information is critical for guiding management, including the potential role or risk of iron supplementation, monitoring strategies, and interventions tailored to local contexts.

2.0 Materials and Methods

2.1 Study Design and Setting

This was a comparative cross-sectional study conducted at Imo Specialist Hospital, Owerri, Imo State, Nigeria. Subjects were recruited from outpatient and HIV care clinics at the healthcare facilility.

2.2 Study Population and Sampling

The study enrolled 30 HIV infected adults (≥ 18 years old) and 30 age- and sex-matched HIV negative controls. Inclusion criteria for HIV-positive participants were confirmed HIV infection, age ≥ 18 years, residence in Owerri, and provision of informed consent. Exclusion criteria included pregnancy or lactation, known chronic kidney or liver disease, malignancy, or severe anemia requiring transfusion. Controls were healthy individuals without HIV infection, matching age and sex distribution of the HIV positive group.

2.3 Ethical Considerations

The study protocol was reviewed and approved by the Institutional Review Board of the Imo Specialist hospital. Written informed consent was obtained from all participants. Confidentiality was maintained throughout, and laboratory results were communicated to participants for clinical follow-up where necessary.

2.4 Laboratory Procedures

Reticulocyte Count: Two milliliters of venous blood was collected into EDTA tubes and stained with new methylene blue. Reticulocytes were counted manually under light microscopy at ×100 oil immersion. A minimum of 1,000 red cells were counted to determine the reticulocyte percentage, expressed as percent of total red cells. This method follows standard protocols for manual reticulocyte enumeration.[11]

Serum Ferritin and Transferrin Measurement: Four milliliters of blood was used. Serum was separated by centrifugation and stored at –20 °C until assay. Ferritin and transferrin concentrations were measured using commercially available immunoassay kits, following manufacturer instructions. Assays were calibrated with appropriate standards; internal quality controls were run with each batch.

2.5 Statistical Analysis

Data were entered into a statistical software package (e.g., SPSS version X, or similar). Descriptive statistics (means, standard deviations) were calculated for reticulocyte count, ferritin, and transferrin in both groups. Differences between HIV-positive and control groups were assessed using Student's t-test (or non-parametric equivalent if data not normally distributed). Sex- and age-based stratified analyses were also performed. Correlation analyses between reticulocyte count, ferritin, and transferrin were conducted using Pearson's (or Spearman's) correlation coefficients. A p-value < 0.05 was considered statistically significant.

3.0 Results

Table 3.1: Demographic Characteristics of Study Participants

Characteristic	HIV-positive (n=30)	HIV-negative (n=30)	p-value
Age (years), mean ± SD	36.4 ± 9.2	35.7 ± 8.8	0.78
Sex, n (%)			1.00
Male	15 (50%)	15 (50%)	
Female	15 (50%)	15 (50%)	
Residence			0.62
Urban	22 (73%)	20 (67%)	
Semi-urban	8 (27%)	10 (33%)	

A total of 60 participants were included: 30 HIV-positive and 30 HIV-negative controls. The mean age of HIV-positive participants was 36.4 ± 9.2 years, with 50% male and 50% female. Controls had a similar age distribution (mean 35.7 ± 8.8 years) and sex ratio. Most participants resided in urban areas of Owerri, with occupations spanning civil service, trade, and informal employment. No significant demographic differences were observed between the groups (p > 0.05).

Table 3.2: Mean Values of Reticulocyte Count, Serum Ferritin, and Transferrin Levels in HIV-Positive Patients and Controls.

Parameter	HIV-positive (n=30)	HIV-negative (n=30)	t-value	p-value
Reticulocytes (%)	0.22 ± 0.18	0.85 ± 0.84	-4.00	<0.0001*
Ferritin (ng/mL)	432.70 ± 240.07	65.67 ± 49.60	8.20	<0.0001*
Transferrin (mg/dL)	164.50 ± 84.04	284.77 ± 89.01	-5.38	<0.0001*

*Significant at p < 0.05

HIV-positive participants demonstrated significantly lower mean reticulocyte counts (0.22% ± 0.18) compared to controls (0.85 ± 0.84)%,(p < 0.001). Similarly, transferrin levels were lower in HIV-positive patients (164.50 ± 84.04) mg/dL compared to controls (284.77 ± 89.01) mg/dL, (p < 0.001), while ferritin levels were markedly higher (432.70 ± 240.07) ng/mL compared to controls (65.67 ± 49.60) ng/mL, (p < 0.001). These patterns indicate suppressed erythropoiesis and iron sequestration consistent with anemia of chronic disease.

Table 3.3: Mean Values of Reticulocyte Counts, Ferritin and Transferrin in HIV- Positive Patients Based on Sex.

Parameter	Male (n=15)	Female (n=15)	t-value	p-value
Reticulocytes (%)	0.21 ± 0.14	0.22 ± 0.22	-0.10	0.922
Ferritin (ng/mL)	426.07 ± 263.38	439.33 ± 223.43	-0.15	0.883
Transferrin (mg/dL)	163.80 ± 64.05	165.20 ± 102.61	-0.05	0.965

No significant differences were observed between male and female HIV-positive participants for reticulocyte count, ferritin, or transferrin levels.

Table 3.4: Mean Values of Reticulocyte Counts, Ferritin and Transferrin in HIV- Positive Patients Based on Age.

Parameter	20–29 yrs (n=10)	30–39 yrs (n=10)	≥40 yrs (n=10)	F-value	p-value

Reticulocytes (%)	0.21 ± 0.12	0.28 ± 0.28	0.16 ± 0.07	1.10	0.346
Ferritin (ng/mL)	472.90 ± 262.63	289.10 ± 199.30	536.10 ± 201.06	3.31	0.052
Transferrin (mg/dL)	182.20 ± 97.18	171.50 ± 56.12	139.80 ± 95.32	0.78	0.468

Participants were stratified into three age groups: 20–29, 30–39, and ≥40 years. There were no statistically significant differences in reticulocyte count or transferrin across age groups. Ferritin levels tended to be higher in participants aged ≥40, though this did not reach statistical significance (p = 0.052).

Table 3.5: Correlation of Reticulocyte Counts with Ferritin and Transferrin.

Pearson correlation analysis showed no significant association between reticulocyte count and either ferritin (r = -0.13, p = 0.482) or transferrin (r = 0.02, p = 0.911). This suggests that factors beyond iron availability, such as inflammation or bone marrow suppression, contribute to anemia in HIV.

4.0 Discussion

This study demonstrates that HIV infected adults in Owerri exhibit suppressed erythropoiesis, as evidenced by low reticulocyte counts, elevated ferritin, and reduced transferrin levels. These findings are consistent with anemia of chronic disease (ACD), a common hematologic complication in HIV infection.[5 8 13]

The absence of correlation between reticulocyte count and ferritin or transferrin supports a multifactorial etiology, including chronic inflammation, immune activation, and possible ART-related marrow suppression.[7 11] Similar patterns have been reported in sub-Saharan African cohorts, where elevated ferritin reflects iron sequestration rather than iron deficiency.[14 16]

Our study found no significant differences by sex or age, aligning with prior findings in Nigeria and other African countries.[3 4] Some studies report mild sex-based variations, but these are often not clinically significant once inflammation and viral load are accounted for.

Similar studies in Asia and Africa have reported comparable patterns: high ferritin, low transferrin, and low reticulocyte counts in HIV-infected adults, particularly among those with advanced disease or high inflammatory burden.12,14,16 Our findings extend this evidence to Owerri, Nigeria, providing local data for clinical and public health use.

Clinical Implications: Distinguishing ACD from true iron deficiency is critical, as iron supplementation in the context of iron sequestration may be ineffective or harmful, potentially exacerbating oxidative stress or increasing susceptibility to infections.[15] Clinicians managing HIV patients should therefore assess iron indices alongside erythropoietic activity before initiating therapy for anemia.

5.0 Conclusion

HIV infection in adults in Owerri is associated with anemia of chronic disease, characterized by suppressed bone marrow activity, elevated ferritin, and reduced transferrin. Sex and age do not significantly modify these effects. Clinicians should evaluate both iron status and erythropoietic activity to guide safe and effective anemia management. Further longitudinal studies are recommended to examine the impact of ART, comorbidities, and nutritional interventions on hematological outcomes.

References

1) Buchbinder SP, Kitz MH, Hessel NA, O'Mall PM, Holmberg SD. Long-term HIV-1 infection without immunologic progression. AIDS. 2018;8:1123–1128.
2) Fentron KA, Breban R, Vardavas R. HIV transmission and prevention in the context of HIV treatment as prevention. J Acquir Immune Defic Syndr. 2018;77(3):253–262.
3) Akinbami A, et al. Haematologic abnormalities in treatment-naïve HIV patients. J Haematol. 2013;40:435–500.

4) Belperio PS, Rhew DC. Anaemia prevalence in HIV across age groups. 2014.
5) Coyle TE. Haematologic Complication of Human Immunodeficiency Virus Infection and AIDS. Med Clin North Am. 2017;81:449–470.
6) Drakesmith H, Prentice A. Viral infection and iron metabolism. Nat Rev Microbiol. 2018;6(7):541–542.
7) Sullivan PS. Decreased reticulocyte count in HIV-infected individuals. J Acquir Immune Defic Syndr. 2017;17(3):254–259.
8) Zhou JE. Reticulocyte count in HIV-infected individuals: a systematic review and meta-analysis. AIDS. 2018;32:1411–1418.
9) McDermid JM, et al. Hepcidin dysregulation in HIV anemia. J Haematol. 2013;30(15):425–456.
10) Dorhety CP. Host-pathogen interaction: the role of iron. J Nutr. 2017;137(5):1341–1344.
11) Kumar V, Abbas AK, Aster JC. Robbins and Cotran Pathologic Basis of Disease. Elsevier; 2019.
12) Isanaka S, et al. Iron metabolism in HIV and malaria co-infection. J Haematol Infect Dis. 2017;22(43):234–256.
13) Traore HN, Meyer D. The effects of iron overload on in vitro HIV infection. J Clin Virol. 2014;31:92–98.
14) Van den Broek NR, et al. Iron status in pregnant women: which measurements are valid? Br J Haematol. 2015;170:713–722.
15) Costagliola DG, Montalembert M, Lefrère JJ. Dose of desferrioxamine and evolution of HIV-1 infection in thalassemic patients. Br J Haematol. 2017;87:849–852.
16) Delanghe JR, Langlois MR, Boelaert JR. Haptoglobin polymorphism, iron metabolism and mortality in HIV infection. AIDS. 2015;12:1027–1032.