Available online at *www.ujpronline.com*
Universal Journal of Pharmaceutical Research
An International Peer Reviewed Journal
ISSN: 2831-5235 (Print); 2456-8058 (Electronic)

RESEARCH ARTICLE

GATA1 PROTEIN AND IRON PROFILE OF HIV-INFECTED SUBJECTS IN UNIVERSITY OF CALABAR TEACHING HOSPITAL, CALABAR, NIGERIA

Enosakhare Asemota[1], Deborah Akpeku[2], Christopher Ogar[1], Osamagbe Aiyudubie Asemota[3], Dennis Akongfe Abunimye[1], Emmanuel Ifeanyi Obeagu[*4,5]

[1]Department of Haematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, University of Calabar, Calabar, Nigeria. [2]Department of Medical Laboratory Science, College of Health Technology, Calabar, Nigeria. [3]Department of Pediatrics, University of Calabar Teaching Hospital, Calabar, Nigeria. . [4]Division of Haematology, Department of Biomedical and Laboratory Science, Africa University, Zimbabwe. [5]Department of Molecular Medicine and Haematology, School of Pathology, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa.

Article Info:

Article History:
Received: 5 February 2026
Reviewed: 8 March 2026
Accepted: 13 April 2026
Published: 15 May 2026

Cite this article:
Asemota E, Akpeku D, Ogar C, Asemota OA, Abunimye DA, Obeagu EI. GATA1 protein and iron profile of HIV-infected subjects in University of Calabar Teaching Hospital, Calabar, Nigeria. Universal Journal of Pharmaceutical Research 2026; 11(2): 25-30.
http://doi.org/10.22270/ujpr.v11i2.1531

***Address for Correspondence:**
Dr. Emmanuel Ifeanyi Obeagu, Department of Biomedical and Laboratory Science, Africa University, Zimbabwe, Tel: +263-77 8025658
E-mail: *emmanuelobeagu@yahoo.com*

Abstract

Background and Aims: HIV infection is associated with alterations in iron metabolism and transcription factors such as GATA1. This study evaluated GATA1 protein, serum iron, total iron-binding capacity (TIBC), and transferrin saturation (TSAT) of HIV-infected subjects in University of Calabar Teaching Hospital, Nigeria.

Subjects and Methods: A total of 90 participants were recruited, comprising 45 HIV-infected subjects on HAART and 45 apparently healthy HIV negative Individuals as controls. Four milliliters of venous blood were collected from each participant and dispensed into plain tubes for serum separation. Serum samples were analyzed for GATA 1 Protein, Iron parameters (Serum Iron (SI), Total iron-binding capacity (TIBC), Unsaturated iron-binding capacity (UIBC), and Transferrin saturation (TSAT) were using Enzyme linked immunosorbent assay (ELISA) and colorimetric methods. Statistical analyses, including Student's t-test, Analysis of Variance and Pearson's correlation were performed using SPSS version 22 with significance set at $p<0.05$.

Results: Serum iron levels were markedly elevated in HIV subjects (183.11±46.59 µg/dl) compared with controls (106.33±25.41 µg/dl; $p<0.001$). Conversely, UIBC (231.11±32.63 µg/dl vs. 327.53±36.76 µg/dl; $p<0.001$) and TIBC (414.13±19.14 µg/dl vs. 433.09±24.01 µg/dl; $p<0.001$) were significantly reduced in HIV subjects. TSAT was substantially higher in HIV-infected participants (43.88± 9.38%) compared to controls (24.64±6.29%; $p<0.001$). GATA1 protein levels showed no significant difference between groups (0.78±1.57 ng/ml vs. 0.69±2.21 ng/ml; $p=0.243$).

Conclusion: HIV-infected individuals on HAART demonstrated significant alterations in iron metabolism, particularly higher serum iron and transferrin saturation, alongside reduced TIBC and UIBC which suggest dysregulation of Iron homeostasis. Although GATA 1 protein level did not differ significantly, these findings suggest that HIV infection may influence erythropoiesis and iron regulation independent of GATA1 protein.

Keywords: Erythropoiesis, ferritin, GATA1, HIV, iron profile, Nigeria.

INTRODUCTION

Human immunodeficiency virus (HIV) infection continues to be a significant global public health issue, especially in sub-Saharan Africa where the disease burden is most severe. Although antiretroviral therapy (ART) is commonly used, individuals living with HIV (PLWH) often encounter hematological issues, with anemia being one of the most prevalent complications. The occurrence of anemia in people infected with HIV is said to vary from 20% to 70%, influenced by disease stage, nutritional condition, and treatment availability[1,2]. Anemia associated with HIV infection has multiple causes, including chronic inflammation, opportunistic infections, nutritional deficits, suppression of bone marrow, and side effects from antiretroviral medication. These elements together interfere with erythropoiesis and iron metabolism, resulting in

changed iron indices and hindered red blood cell production. Iron metabolism is essential for both erythropoiesis and immune functions. In individuals infected with HIV, disruption of iron balance frequently occurs because of ongoing immune activation and the production of inflammatory cytokines. Increased inflammatory mediators boost hepatic hepcidin production, leading to decreased intestinal iron uptake and sequestering iron in macrophages, which ultimately lowers iron availability for erythropoiesis. This process plays a role in the anemia of chronic disease commonly seen in PLWH[3]. Moreover, irregularities in iron parameters like serum iron, ferritin, transferrin saturation, and total iron-binding capacity are frequently observed in HIV infection and may indicate a mix of inflammation, nutritional inadequacies, and disease advancement.

Erythropoiesis is carefully controlled by a system of transcription factors that manage the differentiation and development of erythroid progenitor cells. Among these regulators, GATA binding protein 1 (GATA1) is regarded as a key transcription factor necessary for the development of erythroid and megakaryocytic lineages. GATA1 controls the expression of various genes related to hemoglobin production, heme metabolism, and red blood cell maturation, thus facilitating proper red blood cell development. Changes in GATA1 expression or activity have been linked to inefficient erythropoiesis and various blood disorders, such as anemia and dyserythropoietic conditions[4,5]. The biological function of GATA1 is closely tied to iron and heme metabolism throughout erythroid differentiation. GATA1 controls genes that are crucial for heme biosynthesis and the regulation of metal homeostasis within cells, both of which are vital for hemoglobin production and red blood cell development. Impairment of GATA1 expression has been demonstrated to hinder erythroid differentiation, decrease hemoglobin synthesis, and encourage ineffective erythropoiesis[6]. Considering the significance of iron availability in red blood cell production, changes in iron metabolism linked to HIV infection may affect the regulatory pathways governed by GATA1, thus leading to blood-related issues in affected individuals.

Additionally, HIV infection is known to influence the bone marrow microenvironment and erythroid progenitor cells via persistent inflammation, viral replication, and immune system dysregulation. These mechanisms could disrupt erythropoietic signaling pathways and the transcriptional regulation of genes associated with red blood cell formation. Therefore, assessing molecular regulators like GATA1 in conjunction with biochemical indicators of iron levels may offer greater understanding of the mechanisms that contribute to anemia in individuals infected with HIV. In Nigeria, where the prevalence of HIV continues to be a major health issue, research examining the molecular mechanisms behind HIV-related blood disorders is scarce. This study sought to examine the connection between GATA1 protein and iron levels in HIV-infected individuals receiving care at the University of Calabar Teaching Hospital (UCTH), Calabar, Nigeria. Results from this research could enhance the comprehension of HIV-related anemia and aid in creating specialized diagnostic and treatment approaches for impacted groups.

SUBJECTS AND METHODS

Study area and participants

This study was conducted at the University of Calabar Teaching Hospital (UCTH), situated at No. 10 Ndidem Usang Iso Road, Calabar South, Calabar, Cross River State, Nigeria. Established in 1979, UCTH is geographically positioned at approximately latitude 4.9668° North and longitude 8.3199° East. This location places it within the Calabar Municipality Local Government Area of Cross River State, Nigeria. A total of 90 subjects between the ages of 18 and 60 who gave informed consent were enrolled for the study. Forty-five (45) were HIV infected subjects attending the University of Calabar Teaching Hospital while controls were forty-five (45) apparently healthy non-HIV infected individuals drawn from the general population.

Ethical consideration

This study received ethical approval from the University of Calabar Teaching Hospital Health Research Ethics Committee. Informed consent was obtained from all participants and questionnaire filled before enrollment.

Inclusion criteria

Participants who were included in the study were those who met the following conditions-Adults aged 18 to 60 years, confirmed diagnosis of HIV infection through standard serology method, attending the HIV clinic at the University of Calabar Teaching Hospital (UCTH), Calabar, willing and able to provide informed consent, clinically stable and not requiring emergency care at the time of enrollment. The controls were individuals who were apparently healthy non-HIV infected subjects drawn from the general population, majority of them were students and staff of the University of Calabar and staff of the University of Calabar Teaching Hospital, Calabar who gave consent.

Exclusion criteria

Participants that were below age 18 years, declined to provide informed consent, clinically unstable and emergency patients, HIV infected subjects with known chronic comorbidities that affect iron metabolism or hematological parameters (example chronic kidney disease, liver disease, malignancy, or sickle cell disease) and pregnant or lactating women and individuals who were apparently healthy non-HIV infected subjects who did not give consent.

Sample collection

Four milliliters (4 ml) of venous blood was drawn under aseptic conditions and dispensed into plain tubes for serum separation. Serum samples were analyzed for GATA 1 Protein, Iron parameters (Serum Iron (SI), Total iron-binding capacity (TIBC), Unsaturated iron-binding capacity (UIBC), and Transferrin saturation (TSAT).

Laboratory analysis

GATA 1 Protein, Iron parameters (Serum Iron (SI), Total iron-binding capacity (TIBC), Unsaturated iron-binding capacity (UIBC), and Transferrin saturation (TSAT) using Enzyme Linked Immunosorbent Assay (ELISA) and colorimetric methods.

Statistical analysis

Data analysis was done using the statistical package for social sciences (SPSS version 24). Chi-square (χ^2) test was used for comparison of demographic data, Student's t-test was used to test for mean difference between groups (HIV subjects and Controls) and Analysis of Variance was used to determine variations among group means based on duration of Highly Active Anti-retroviral therapy (HAART) intake. A probability value $p<0.05$ was considered statistically significant.

Table 1: Demographic distribution of the study population.

Parameters	Category	HIV Subjects (n=45)	Control (n=45)	χ^2 Coefficient	*p*-value
Age (Years)	<40 years	n=16 (36%)	n=36 (80%)	87.912	<0.05
	≥40 years	n=29 (64%)	n=9 (20%)		
Education	SSCE	n=8 (18%)	n=3 (7%)		
	Bachelor's Degree	n=33 (74%)	n=18 (40%)		
	National Diploma	n=2 (4%)	n=18 (40%)	87.96	<0.05
	Master's Degree	n=2 (4%)	n=6 (13%)		
Gender	Male	n=13 (29%)	n=15 (33%)		
	Female	n=32 (71%)	n=30 (67%)	45.4	<0.05
Marital Status	Married	n=29 (64%)	n=20 (44%)		
	Not Married	n=16 (36%)	n=25 (56%)	52.22	<0.05
Occupation	Business	n=21 (47%)	n=10 (22%)		
	Civil Servant	n=6 (13%)	n=16 (36%)		
	House Wife	n=7 (16%)	n=0 (0%)		
	Retiree	n=1 (2%)	n=0 (0%)	78.74	<0.05
	Students	n=10 (22%)	n=18 (40%)		
	Market Women	n=0 (0%)	n=1 (2%)		

RESULTS

Table 1 shows the demographic distribution of the study population on both HIV-infected subjects and control. A total of 90 participants were enrolled, comprising 45 HIV-infected subjects and 45 controls. With respect to age, 16 (36%) of the HIV-infected subjects were aged below 40 years, whereas 29 (64%) were aged 40 years and above. In contrast, among the control group, 36 (80%) were younger than 40 years and only 9 (20%) were aged 40 years and above. The difference in age distribution between the two groups was statistically significant ($\chi^2=87.912$, $p<0.05$). In terms of educational attainment, the majority of HIV-infected participants (33; 74%) held a Bachelor's degree, 8 (18%) had a Senior Secondary Certificate (SSCE), 2 (4%) had a National Diploma, and 2 (4%) possessed a Master's degree. Among the controls, 18 (40%) had a Bachelor's degree, 3 (7%) had an SSCE, 18 (40%) had a National Diploma, and 6 (13%) had a Master's degree. This difference was statistically significant ($\chi^2=87.96$, $p<0.05$). Regarding gender, 13 (29%) of the HIV-infected subjects were male while 32 (71%) were female. Among the controls, 15 (33%) were male and 30 (67%) were female, and this gender distribution also showed a significant difference ($\chi^2=45.4$, $p<0.05$). With respect to marital status, 29 (64%) of the HIV-infected participants were married and 16 (36%) were not married. In comparison, 20 (44%) of the controls were married while 25 (56%) were unmarried. The variation between both groups was statistically significant ($\chi^2=52.22$, $p<0.05$). The occupational distribution revealed that among HIV-infected subjects, 21 (47%) were business persons, 6 (13%) were civil servants, 7 (16%) were housewives, 1 (2%) was a retiree, and 10 (22%) were students. In contrast, among the control group, 10 (22%) were business persons, 16 (36%) were civil servants, none were housewives or retirees, 18 (40%) were students, and 1 (2%) was a market woman. This difference in occupational distribution between the two groups was statistically significant ($\chi^2=78.74$, $p<0.05$). Table 2 shows Serum iron levels were markedly elevated in HIV subjects (183.11±46.59 µg/dl) compared with controls (106.33±25.41 µg/dl; $p<0.001$).

Conversely, UIBC (231.11±32.63 µg/dl vs. 327.53± 36.76 µg/dl; $p<0.001$) and TIBC (414.13±19.14 µg/dl vs. 433.09±24.01 µg/dl; $p<0.001$) were significantly reduced in HIV subjects.

Table 2: GATA 1 protein and serum iron parameters of HIV infected subjects and controls.

Parameters	HIV Subjects (n=45)	Control (n=45)	T - value	*p*-value
GATA 1 (ng/mL) (0.625-4ng/mL)	0.78±1.57	0.69±2.21	1.058	0.243
IRON(ug/dl) (65–175 µg/dL)	183.11±46.59	106.33±25.41	9.704	<0.001
UIBC(ug/dl) (150–375 µg/dL)	231.11±32.63	327.53±36.76	13.159	<0.001
TIBC(µmol/L) (45–72 µmol/L)	414.13±19.14	433.09±24.01	4.141	<0.001
TSAT(%) (20–50%)	43.88±9.38	24.64±6.29	11.428	<0.001

Table 3: GATA 1 protein and serum iron parameters of HIV infected subjects based on HARRT intake.

Parameters	1-3 Years (n=16)	>3-5 Years (n=17)	>5-7 Years (n=12)	*p*-value
GATA 1 (ng/mL) (0.625–4ng/mL)	0.88±1.61	1.00±2.03	0.35±0.19	0.532
IRON(ug/dl) (65–175 µg/dL)	184.00±44.65	188.00±55.47	175.00±37.16	0.762
UIBC(ug/dl) (150 –375µg/dL)	233.56±31.11	224.58±37.33	227.18±28.25	0.567
TIBC(µmol/L) (45–72 µmol/L)	417.31±19.97	412.59±21.06	412.08±15.87	0.717
TSAT (%) (20–50%)	43.81±8.94	45.07±11.11	42.29±7.67	0.743

TSAT was substantially higher in HIV-infected participants (43.88±9.38%) compared to controls (24.64± 6.29%, $p<0.001$). GATA1 protein levels showed no significant difference between groups (0.78±1.57 ng/ml vs. 0.69±2.21 ng/ml, $p=0.243$,

DISCUSSION

This research assessed the GATA1 protein levels and iron profile indicators in HIV-positive individuals visiting the University of Calabar Teaching Hospital, Calabar, Nigeria, and contrasted them with seemingly healthy controls. The results indicated notable changes in iron metabolism in individuals infected with HIV, whereas GATA1 protein levels did not reveal a statistically significant variation between the two groups. These findings offer understanding of the intricate relationship among HIV infection, iron metabolism, and the regulation of erythropoiesis. The demographic traits of the study population displayed notable variations between HIV-infected individuals and controls regarding age, education level, gender, marital status, and profession. The larger percentage of participants aged ≥40 years among HIV-infected individuals may indicate the long-term aspect of HIV infection and enhanced longevity resulting from the broad accessibility of highly active antiretroviral therapy (HAART). Comparable demographic trends have been noted in research showing that HIV prevalence is often greater among adults during their economically active years[7,8]. Furthermore, the increased percentage of female participants among HIV-infected individuals noted in this study corresponds with findings indicating that women in sub-Saharan Africa face a disproportionate risk of HIV infection due to biological, socio-economic, and cultural influences[9-11]. This study's findings showed notably higher serum iron levels in HIV-infected individuals than in the control group. This discovery indicates a disruption in iron balance linked to HIV infection. Increased serum iron levels can arise from chronic inflammation, heightened iron release from macrophages, and dysfunctional regulation of proteins involved in iron storage and transport.

Table 4: GATA 1 protein and serum iron parameters of HIV infected subjects based on gender.

Parameters	Females (n=32)	Males (n=13)	*p*-value
GATA 1 (ng/mL) (0.625-4 ng/mL)	0.72±1.50	0.96±11.78	0.646
IRON (ug/dl) (65–175 µg/dL)	186.38±49.95	175.07±37.65	0.467
UIBC (ug/dl) (150–375 µg/dL)	229.87±35.14	234.16±26.47	0.695
TIBC (µmol/L) (45–72 µmol/L)	416.25±20.71	408.92±13.91	0.249
TSAT (%) (20–50%)	44.40±10.03	42.60±7.79	0.565

Past research has shown that HIV infection may change iron metabolism via inflammatory pathways that involve cytokines like interleukin-6, which regulate hepcidin production and affect systemic iron distribution[12-14]. Greater iron availability may also boost viral replication, since iron is a crucial cofactor for various viral enzymes that play a role in HIV replication. In comparison, unsaturated iron-binding capacity (UIBC) and total iron-binding capacity (TIBC) were markedly reduced in HIV-infected individuals versus controls. Lowered UIBC and TIBC may indicate decreased transferrin production resulting from chronic inflammation or liver impairment frequently seen in those with prolonged HIV infection. Comparable decreases in iron-binding capacity have been reported in anemia associated with HIV and inflammatory states, where transferrin levels drop as a component of the acute phase response[15,16]. The current study revealed notably elevated transferrin saturation (TSAT) in HIV-infected individuals relative to controls. Increased TSAT reflects a higher percentage of transferrin in circulation associated with iron, which could imply enhanced iron availability or compromised regulation of iron transport processes. Elevated transferrin saturation has been linked to oxidative stress and excessive tissue iron, factors that could worsen disease progression in individuals infected with HIV[17].

Table 5: GATA 1 protein and serum iron parameters of HIV infected subjects based on age.

Parameters	≤ 44 Yrs (n=25)	> 44 Yrs (n=20)	*p*-value
GATA 1 (ng/mL) (0.625-4ng/mL)	0.79±1.42	0.79±1.79	0.999
IRON(ug/dl)(65–175 µg/dL)	179.80±45.90	187.25±48.31	0.600
UIBC(ug/dl) (150–375 µg/dL)	232.84±32.85	228.95±33.07	0.696
TIBC(µmol/L) (45–72 µmol/L)	412.48±17.27	416.20±21.52	0.523
TSAT(%) (20–50%)	43.28±9.42	44.63±9.52	0.640

These changes in iron levels emphasize how chronic infection and inflammation affect iron metabolism in populations infected with HIV. Even with the substantial changes noted in iron parameters, the levels of GATA1 protein showed no significant difference between individuals infected with HIV and control subjects. GATA1 is an essential transcription factor that plays a key role in erythroid differentiation and maturation. The lack of a notable difference might suggest that HIV infection does not directly influence circulating GATA1 protein levels, or that HIV's impact on erythropoiesis happens through alternative regulatory mechanisms like inflammatory cytokines or suppression of bone marrow. Previous studies indicate that GATA1 mainly operates in erythroid precursor cells in the bone marrow, and the levels of circulating protein may not completely represent intracellular transcriptional activity[18]. Consequently, the absence of notable change seen in this study could be attributed to the compartmentalized characteristics of GATA1 expression.

Further analysis of HIV-infected subjects based on duration of HAART intake revealed no significant differences in GATA1 protein levels or iron parameters among participants receiving treatment for 1–3 years, >3–5 years, or >5–7 years. This finding suggests that long-term HAART exposure may not significantly influence iron metabolism or GATA1 expression in this study population. Some studies have reported that antiretroviral therapy may gradually improve hematological abnormalities by suppressing viral replication and reducing chronic inflammation. However, the absence of significant differences in the present study may indicate that factors other than treatment duration such as nutritional status, co-infections, or baseline disease severity may play more important roles in determining iron metabolism among HIV-infected individuals[17,18]. Similarly, when the HIV-infected subjects were stratified according to gender and age, no statistically significant differences were observed in GATA1 protein levels or iron profile parameters. This suggests that the alterations in iron metabolism observed in this study may be primarily related to HIV infection itself rather than demographic characteristics such as age or sex. Previous studies have also indicated that while gender and age can influence iron metabolism under normal physiological conditions, the impact of chronic infections such as HIV may overshadow these differences due to persistent immune activation and inflammatory processes[17].

CONCLUSIONS

In conclusion, HIV infection among those on HAART is linked to a unique pattern of iron imbalance, marked by notably increased serum iron and transferrin saturation, as well as decreased TIBC and UIBC. These changes align with impaired iron balance and may indicate modifications in iron use, storage, or transport amid chronic infection and therapy. Even with these notable biochemical changes, GATA1 protein levels remained similar between HIV-infected individuals and controls, indicating that the identified disruptions in iron metabolism and possible impacts on erythropoiesis may happen via mechanisms that do not involve GATA1. Together, these results emphasize the necessity for more careful observation of iron levels in HIV-positive individuals receiving HAART and stress the significance of investigating different regulatory pathways that connect HIV infection, iron metabolism, and blood cell production.

ACKNOWLEDGEMENTS

The authors would like to acknowledge all researchers whose work contributed to this work.

AUTHOR'S CONTRIBUTIONS

ObAsemota E: formal analysis, conceptualization, writing original draft. **Akpeku D:** conceptualization, data organization, supervision. **Ogar C:** conceptualization, data organization, supervision. **Asemota OA:** critical review. **Abunimye DA:** data organization. **Obeagu EI:** editing, critical review. Final manuscript was checked and approved by all authors.

DATA AVAILABILITY

The empirical data used to support the study's conclusions are available upon request from the corresponding author.

CONFLICT OF INTEREST

None to declare.

REFERENCES

1. Andounimye AD, Asemota EA, Akpan UO. Haematological parameters of HIV infected subjects attending University of Calabar Teaching Hospital, Calabar, Nigeria. Nig J Pure Appl Sci 2025; 38:2. *https://doi.org/10.48198/NJPAS/25.A34*
2. Liao R, Bresnick EH. Endogenous small molecule effectors in GATA transcription factor mechanisms governing biological and pathological processes. Exp Hematol 2024; 137:104252. *https://doi.org/10.1016/j.exphem.2024.104252*
3. Lopez GH, Sarri ME, Flower RL, *et al*. Impact of transcription factors KLF1 and GATA1 on red blood cell antigen expression: A review. Immunohematol 2024;40(1):1-9. *https://doi.org/10.2478/immunohematology-2024-002*
4. El Hoss S, Shangaris P, Brewin J, *et al*. Reduced GATA1 levels are associated with ineffective erythropoiesis in sickle cell anemia. Haematologica 2025; 110(5):1150-1163. *https://doi.org/10.3324/haematol.2024.286010*
5. Obeagu EI, Obeagu GU, Ukibe NR, Oyebadejo SA. Anemia, iron, and HIV: Decoding the interconnected pathways: A review. Medicine (Baltimore) 2024;103(2): e36937. *https://doi.org/10.1097/MD.0000000000036937*
6. Akpan PA, Asemota EA, Etura JE. Assessment of markers of platelet immune activation in malaria, tuberculosis and human immunodeficiency virus infection. African J Clin Exper Microbiol 2025; 26:3. *https://doi.org/10.4314/ajcem.v26i3.7*
7. World Health Organization. HIV infection and antiretroviral therapy guidelines. WHO Press 2022.
8. Chang HC, Bayeva M, Taiwo B, *et al*. Short communication: High cellular iron levels are associated with increased HIV infection and replication. AIDS Res Hum Retroviruses 2015 Mar; 31(3):305-12. *https://doi.org/10.1089/aid.2014.0169*

9. Obeagu EI, Okafor CJ. Intersecting Pathways: The complex relationship between HIV and fertility in women with sickle cell disease. Universal J Pharm Res 2025; 10(6): 98-105. *http://doi.org/10.22270/ujpr.v10i6.1468*
10. Gonçalves JL, Silva MCA, Roma EH, *et al*. Iron intake is positively associated with viral load in antiretroviral naïve Brazilian men living with HIV. Mem Inst Oswaldo Cruz 2020;114: e190350. *https://doi.org/10.1590/0074-02760190350*
11. Obirikorang C, Issahaku RG, Osakunor DN, *et al*. Anaemia and iron homeostasis in a cohort of HIV-infected patients: A cross-sectional study in Ghana. AIDS Res Treat 2016; 2016:1623094. *https://doi.org/10.1155/2016/1623094*
12. Frosch AEP, Ayodo G, Odhiambo EO, *et al*. Iron deficiency is prevalent among HIV-Infected Kenyan adults and is better measured by soluble transferrin receptor than ferritin. Am J Trop Med Hyg 2018;99(2):439-444. *https://doi.org/10.4269/ajtmh.18-0208*
13. Gebrehiwot GT, Tilahun M, Gebreyohannes G, *et al*. A retrospective study on HIV virological recovery patterns and factors associated with HIV viral treatment using highly active antiretroviral therapy (HAART) in public health facilities in tigray, Northern Ethiopia. Virol J 2025;22(1):317. *https://doi.org/10.1186/s12985-025-02828-1*
14. Wagnew F, Eshetie S, Alebel A, *et al*. Burden of anemia and its association with HAART in HIV infected children in Ethiopia: A systematic review and meta-analysis. BMC Infect Dis 2019;19(1):1032. *https://doi.org/10.1186/s12879-019-4656-1*
15. Khan NH, Verma C, Beg MMA, *et al*. Evolution of hematobiochemical profiles in newly diagnosed HIV patients and HIV-TB co-infected patients: Correlation with immunological and virological status. Immunotargets Ther 2024; 13:691-705. *https://doi.org/10.2147/ITT.S495295*
16. Obeagu EI. Reprogramming iron metabolism in HIV: Molecular mechanisms driving viral persistence and disease progression. Universal J Pharm Res 2025; 10(5): 99-109. *http://doi.org/10.22270/ujpr.v10i5.1431*
17. Obeagu. Beyond HIV-associated anemia: Exploring the consequences of repeated blood transfusions in HIV care. Universal J Pharm Res 2025; 10(4): 84-95. *http://doi.org/10.22270/ujpr.v10i4.1399*
18. Obeagu EI, Okafor CJ. Immune aging in the young: Consequences of HIV-Induced senescence in children. Universal J Pharm Res 2026; 11(1): 60-66. *http://doi.org/10.22270/ujpr.v11i1.1491*