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Full Length Research Article

PREVALENCE OF HIV-1 GENETIC SUBTYPES IN ANTIRETROVIRAL DRUG RESISTANCE PATIENTS IN MINNA, METROPOLIS, NIGER STATE, NIGERIA

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ABSTRACT
The genetic diversity of HIV-1 significantly influences the clinical management of HIV infection, particularly regarding antiretroviral therapy (ART) efficacy and drug resistance. This study investigates the incidence and distribution of HIV-1 genetic subtypes among ART-experienced individuals exhibiting drug resistance in Minna Metropolis, Niger State, Nigeria. A total of 120 ART-experienced HIV-positive patients with suspected virologic failure were enrolled. Plasma samples were collected, and viral RNA was extracted and amplified for the pol gene region. Phylogenetic analysis was conducted to determine subtype distribution, while genotypic resistance testing identified resistance-associated mutations. Results indicated a predominance of CRF02_AG (58.3%), followed by subtype G (25.0%), subtype A1 (10.0%), and recombinant forms (6.7%). Resistance mutations were found in 73.3% of patients, with common mutations including M184V/I, K103N, and Y181C. The findings highlight the urgent need for tailored ART regimens and continued molecular surveillance to optimize treatment outcomes in the region.

Keywords: HIV-1, Genetic Subtypes, RF02_AG, Drug Resistance Mutations, HIV Diversity, Nigeria

INTRODUCTION

The major defining characteristic of HIV-1 is its high genetic diversity, which is a result of a fast replication cycle, high mutation rate, and high recombination rates (UNAIDS, 2023). This high genetic diversity results in the presence of different variants of the virus in different regions of the world, and the emergence of new variants, especially in areas with multiple circulating subtypes continues to occur (Adhiambo *et al.,* 2021). There are four phylogenetic groups of HIV-1: M (major), O (outlier), N (non-M/non-O), and the most recent group P. Group M is responsible for the vast majority of global HIV-1 infections, accounting for more than 90% of cases worldwide and driving the global AIDS pandemic. Group M is further subdivided into nine subtypes: A, B, C, D, F, G, H, J, and K. within these, subtype A is divided into six sub-subtypes (A1, A2, A3, A4, A5, and A6), and subtype F into two sub-subtypes (F1 and F2). Additionally, over 102 circulating recombinant forms (CRFs) and around 100 unique recombinant forms (URFs) have been identified, reflecting the extensive genetic diversity and recombination events characteristic of HIV-1 group M. This diversity contributes to the complex epidemiology and ongoing evolution of the virus globally (Buonaguro *et al*., 2007; Giovanetti *et al*, 2020). Subtype B is the predominant strain of HIV-1 in high-income countries whereas in low-income and middle-income countries (LMICs), especially in the African continent, the non-B subtypes together with several CRFs and URFs currently drive the epidemic. In sub-Saharan Africa, several studies have reported the presence of multiple HIV-1 subtypes along with a number of CRFs, such as CRF01_AE in Central Africa and CRF02_AG in West Africa (Nazziwa *et al,* 2020). Macrophage activation and secretion of pro-inflammatory cytokines and chemokines, such as IL-10, caused by HIV infection might have a significant impact on HIV pathogenesis (Kruize, 2019). Until recently, antiretroviral drugs were developed and tested for efficacy with subtype B viruses as the reference. The clinical effectiveness of these drugs and their patterns of drug resistance were established in clinical trials conducted for the most part on patients infected with subtype B (Kosakovsky & Smith, 2009). Although most drugs would be expected to act on targets that were conserved as a result of their critical function in the viral replication cycle such as reverse transcription, protein processing (protease), integration, and coreceptor binding, it seems probable that genetic differences between subtypes might impact the drug resistance pathways or kinetics of drug resistance development (Chaplin *et al*., 2011). It has been shown that the mutational pathways to drug resistance to nucleoside reverse transcriptase inhibitor (NRTI) drugs may vary among different HIV-1 subtypes (Nastri *et al*., 2023). In addition, non-B subtype HIV genomes carry subtype-specific polymorphisms that act as minor mutations in subtype B, particularly in the protease gene. The impact of these genetic differences on the clinical response to antiretroviral therapy has yet to be fully assessed (Dumans *et al*, 2009). Globally, the management of HIV infection has been seriously affected by the genetic diversity of the virus, because of the emergence of drug-resistant variants and resultant treatment failure. A 2017 report from the WHO revealed that in quite a number of LMICs, about 10% of HIV-infected patients initiating antiretroviral therapy (ART) have pre-existing HIV drug resistance to efavirenz and nevirapine. (WHO, 2017). These pre-treatment drug resistance mutations can result in poor treatment outcomes and increased rate of death in adults and children. The Human Immunodeficiency Virus (HIV) response in Nigeria has reached roughly 1 million patients with

Highly-Active Antiretroviral Therapy (HAART) in about 1500 treatment sites (NACA, 2019). Despite the increasing access to antiretroviral medications several studies have demonstrated suboptimal levels of viral load suppression among different population groups especially in low resource settings (Kobabchhim *et al*, 2019). Achieving the UNAIDS 90-90-90 targets by 2020—and the subsequent 95-95-95 targets by 2030—relies critically on ensuring sustained viral suppression in people living with HIV who are on ART. The 90-90-90 strategy aims for 90% of all people living with HIV to know their status, 90% of those diagnosed to receive sustained ART, and 90% of those on treatment to achieve viral suppression, which translates to at least 73% of all people living with HIV having suppressed viral loads. This viral suppression is essential not only for maintaining individual health but also for preventing onward transmission of HIV, thereby contributing to epidemic control (Bain *et al*., 2017; NACA, 2019). Despite significant progress—by the end of 2019, approximately 81% knew their status, 67% were on treatment, and 59% had viral suppression globally—the world fell short of the 90-90-90 targets, partly due to challenges such as treatment interruptions, access disparities, and the COVID-19 pandemic's impact on healthcare delivery (UNAIDS, 2020). Sustained viral suppression requires continuous access to quality ART, adherence support, and viral load monitoring to detect and address treatment failure early. The 95-95-95 targets set for 2030 build on this foundation, aiming for even higher levels of diagnosis, treatment coverage, and viral suppression to end the AIDS epidemic. Achieving these goals demands intensified efforts to close gaps in testing, treatment initiation, retention, and adherence, particularly among key populations and underserved groups, ensuring equitable access to HIV services worldwide (Bain *et al.,* 2017; UNAIDS, 2020). Increasing genotypic HIV drug resistance and immune reactivation inevitably occurs after a period of ART. The genetic mutations of HIV strains could influence the therapy response to different medication and the occurrence of treatment failure. (Kakubu *et al*., 2022). Therefore, it is necessary to identify the drug resistance-related mutations of HIV genotypes. This study seeks to detect the demographic distribution and clinical staging of recent HIV-1 infections, evaluate immune responses, characterize subtype diversity and determine the presence of drug resistance mutations among subtypes from a cohort of PLWH in Minna Niger State, North Central Nigeria. In HIV-1 infection, inflammatory responses play a crucial role in the pathogenesis of the disease, even when patients are receiving ART. Human immunodeficiency virus type 1 (HIV-1) is characterized by its high genetic variability, resulting in multiple subtypes and CRFs. This diversity significantly impacts treatment outcomes, viral pathogenicity, and resistance development. In sub-Saharan Africa, including Nigeria, CRF02_AG and subtype G predominate, but the prevalence of other subtypes and recombinants is increasing. Minna, the capital of Niger State, is experiencing a growing HIV burden, with ART scale-up and emerging challenges of drug resistance. Despite national efforts in viral load monitoring, limited data exist on HIV-1 subtype distribution and drug resistance patterns in Minna Metropolis. This study aims to fill that gap by analyzing the incidence of HIV-1 subtypes and associated drug resistance mutations in ART-experienced patients with virologic failure. This study aimed to detect the demographic distribution and clinical staging of recent HIV-1 infections,

MATERIALS AND METHODS

Study Setting

Nigeria is located in sub-Sahara Africa with a population of over 200 million People occupying a landmass of 923,768 square kilometers and 1.4% current national prevalence of HIV infections. (Niger State Government, 2017). The projected population for Niger State for 2024 is 6,783,300 million (Wikipedia, 2024). The State HIV prevalence currently stands at 0.7% (Daily Post Nigeria, 2025). This study was conducted at Ibrahim Badamasi Babangida Specialist Hospital (IBBSH), an ultra-modern tertiary hospital wholly owned by Niger State Government and a major referral centre in Niger State, located about 10 km from Paiko town. It was established and commissioned in the 21/08/1993 by the then military head of state, General Ibrahim Badamosi Babangida. IBBSH is a 100- bed capacity hospital, providing clinical service (including a comprehensive up to date Laboratory services), training of medical personnel and research in medicine, a functional ART clinic caring for about 900 HAART patients and active ART team comprising of Medical Doctors, Medical Laboratory Scientists, Pharmacists, and Triad Nurses in addition to other clinical services caring for PLWHA since 2010. The ART clinic and Laboratory services is being supported by Center for Clinical Care and Research Nigeria (CCCRN) (A non-Governmental Organization) as the implementing partners that provides health care services for HIV infected patients and the National TB/Leprosy control program supported by Global fund provide health care services for TB patients and has a reference Multi-Resistant MTB Laboratory. The ART services of the centre have been rated on several occasions as one of the best in the North-Central Nigeria by the implementing partners.

Study Design

This descriptive, cross-sectional study evaluated HIV-1 infected patients attending the ART Clinic/Laboratory of IBB Specialist Hospital in Minna, Niger State, Nigeria. Participants included adults (≥18 years) that had been on first-line HAART regimens for ≥6 months at enrolment. The study spanned January 2024 to December 2024. The primary objective was to define virological failure using two consecutive viral load measurements >1000 copies/mL. Additional parameters included immunological (CD4 counts), inflammatory (hs-CRP, IL-6, IL-10, TNF-α), and virological markers (HIV-1 Viral Load and genetic subtypes), Participants underwent face-to-face interviews using structured questionnaires capturing: Socio-demographics (Age, sex, education, employment, and geographical location), Treatment history (Adherence patterns and ART duration), HIV knowledge: (Transmission routes, symptoms, prevention, and information sources), Health practices (Risk behaviours and healthcare engagement). Stratification was done by Virological status (Viral load trajectories and failure patterns). Immunological markers (CD4+ T-lymphocyte counts), inflammatory profiles (hs-CRP, IL-6, IL-10, and TNF-α levels) and Viral genetics (HIV-1 subtypes and treatment outcomes).

Ethical Consideration

This study adhered to Nigerian HIV research ethics standards, incorporating community-aligned protocols and rigorous oversight. Ethical approval was secured from the Health Research Ethics Committee (HREC) of IBB Specialist Hospital Minna (certificate No. IBBSH/SUB/615/M2023-07), with additional alignment to Nigeria's National HIV Research Policy requirements for community-inclusive design. Participants received culturally

adapted explanations of the study's purpose, risks, and benefits in their preferred language, addressing literacy barriers through verbal translations. The process met Helsinki Declaration standards (World Medical Association, 2024). and Nigeria's emphasis on local-language comprehension (Ogunrin *et al.,* 2013) Data and records were stripped of personal identifiers, using unique codes for linkage and withdrawal rights explicitly guaranteed without service penalties. The HREC conducted protocol-driven monitoring to ensure adherence to approved procedures, including verification of consent, documentation, integrity and participant recruitment fairness—key measures to mitigate risks identified in Nigerian HIV research audits

Study Population
The study population involved all the HIV-infected clients attending the ART clinic that have satisfied the eligibility criteria and consented to participate in the study within a time bound of 6 months. This is because the clients are usually given 2-3 months appointment for a refill of their medication and clinical visit. This would avail the clients within the study population the opportunity to participate in the study thereby limiting the chance of repetition. Enrolled subjects were monitored, followed up and observed for immune responses and adherence to Anti-retroviral drugs.

Sample Size Estimation.
The minimum sample size (n) was calculated by single proportion formula based on 2.5% estimated Virological Failure on HAART subjects in Niger State (NACA, 2022).
This will be determined by $n = \frac{Z^2pq}{d^2}$ (Pourhoseingholi *et al*, 2013)
n = Desired sample size
z = Standard Normal Deviation given as 1.96 with 95% confidence level
p = Estimated proportion of the population with the estimated Virological Failure on HAART (2.5%). (NACA, 2022)
q = 1 – p
d = Margin of error/Degree of accuracy given as 0.05 (precision)
$Z^2 = 1.96^2 = 3.8416$
P = 2.5% = 0.025
q = 1 – 0.025 = 0.975
$d^2 = 0.05^2 = 0.0025$
n = 3.8416 x 0.025 x 0.975 x 0.0025 = 37
A minimum of 37 participants was estimated to be sufficient in representing the population at 5% error margin and 95% confidence interval with an addition of 15% for response rate (Pourhoseingholi *et al*., 2013). A total of 43 participants were used in this study.

Duration of Study
The targeted period of time between the commencements of collection of samples through analysis to statistical analysis was twelve (12) months.

Inclusion Criteria
HIV-1 HAART-treated patients who have experienced virological failure within 6 months (VF: repeated VL = > 1000 copies/mL).
HIV-1 HAART-treated patients above 18 years
HIV- HAART-treated patients with or without HBV, HCV and TB co-morbidities

Exclusion Criteria
HAART-treated patients who have not experienced virological failure within 6 months (VF: repeated VL = > 1000 copies/mL), with HAART-treated patients below 18 years.

Data Collection Instruments
Structured Questionnaires: Structured data collection form was designed, demography information, medical history and the clinical characteristics/outcomes data, Environmental/Host related exposures were obtained from patient's hospital folders. The data were then transferred into the data collection form. Acceptability was determined using some set of questions adapted from a similar study (Oluwalana *et al*., 2022).

Sample Collection, Transportation and Storage: Blood and Sputum samples were collected based on standard operating procedures. Serum and Plasma samples were obtained from whole blood based on standard operating procedures.
Every participant was assigned a unique and confidential identifier code for questionnaire and samples. Nine milliliters (9ml) of blood was collected from each participant in an ethylenediamine tetraamine acid (EDTA) anticoagulated vacutainer tube by a trained phlebotomist. One milliliter (1ml) of the well mixed whole blood was transferred to a 2ml-container for CD4 cells count. The remaining 8ml of blood was centrifuged at 3000 rpm for 20 minutes and the plasma was transferred using clean Pasteur pipette into two cryovials for HIV-1 plasma viral load determination, RT-PCR, HIV-1 genetic sub-types sequencing and sequencing of protease and reverse transcriptase region in the *pol* gene. The CD4 cells count was conducted in the hospital where the samples were collected. Another five milliliters (5ml) of blood was collected from each participant in a plain vacutainer tube by a trained phlebotomist. The 5ml blood was centrifuged at 3000 rpm for 20 minutes after clotting and the serum was transferred using clean Pasteur pipette into two cryovials for IL-6, IL-10, hs-CRP, HBV and TNF-α, HCV for analysis in Immunology Laboratory of Department of Medical Laboratory Services, IBB Specialist Hospital, Minna where the samples were collected. Plasma samples were stored at -20°C for further molecular analysis. All stored samples were transported to Clinical Virology/Molecular Laboratory, University College Hospital (UCH) Ibadan for RT-PCR, HIV-1 sub-types analysis and sequencing of protease and reverse transcriptase region in the *pol* gene for drug resistance testing and Defense Reference Laboratory Mogodishu Cantonment Abuja FCT for HIV-1 plasma viral load determination.

Analytical Procedure
Detection of HIV-1 Using Rapid Diagnostic Test Kit
Plasma samples of the study participants that were previously tested positive by the National testing algorithm kits (Determine™ HIV-1/2, USA; Uni-Gold™, Ireland; HIV-1/2 STAT-PAK®, USA) were tested using MULTISURE® HIV Rapid Test (MP Biomedicals, Germany). MULTISURE® HIV Rapid Test (MP Biomedicals, Germany) was used to detect HIV type in participants' plasma. Kit and samples were brought to room temperature before testing. The test device was labelled with the sample number and 25µl plasma was added into the kit's square well (Figure 1). When the sample front reached the blue indicator line, 3 drops of Chase Buffer were added into the kit's oval well. The "HIV" Pull Tab was pulled out until resistance was felt. One drop of Chase Buffer was added into the square well and result was read after 20 minutes. Results were

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interpreted as presented in Figure 2.

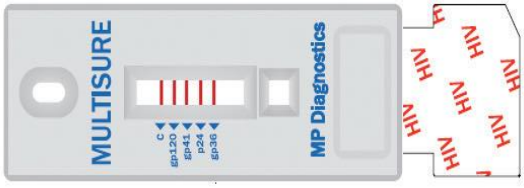

Figure 1: Multsure, test device

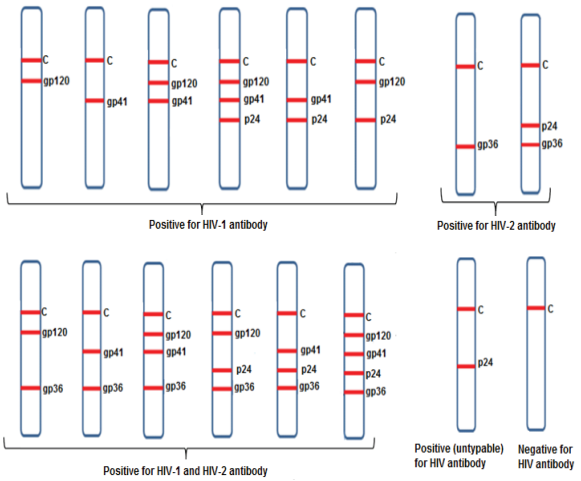

Figure 2: Interpretation of the test result

CD4 Cell Estimation

The CD4+ T-cell count was carried out using the VISITECT® CD4 Advanced Disease Rapid test kit. The VISITECT® CD4 Advanced Disease Rapid Test is an immunochromatographic assay designed to estimate CD4 protein levels associated with CD4+ T cells in human whole blood. This test is particularly useful for identifying patients with advanced HIV disease, as it provides rapid results at the point of care.

Principle

The VISITECT CD4 test operates on the principle of immunochromatography, which involves the use of antibodies to detect specific proteins—in this case, CD4 proteins associated with CD4+ T cells. A sample of whole blood (either capillary or EDTA venous) is added directly to the test device. The red blood cells and monocytes are retained in the blood collection pad, while other white blood cells, including CD4+ T cells, migrate to the reaction area. After the addition of a buffer solution, lysis of the white blood cells occurs, releasing full-length CD4 proteins into the test strip. The test strip contains a line of capture monoclonal antibodies (MAb) specific for the cytoplasmic domain of CD4. These antibodies bind to the released CD4 proteins, forming a complex. Colloidal gold-labelled antibodies against CD4 bind to the captured CD4 proteins, leading to the formation of a visible test line on the strip. The intensity of this line correlates with the amount of CD4 protein present in the sample. A reference line (200 line) is included on the test device, allowing for the estimation of CD4 levels by comparison to a predetermined cut-off that corresponds to a CD4 count of 200 cells/µL. The presence of a test line indicates the level of CD4 proteins; If the test line is stronger than the reference line, the CD4 count is above 200 cells/µL. If the test line is equal to or weaker than the reference line, the CD4 count is below 200 cells/µL, indicating advanced HIV disease. For the test result to be valid, both the control line and the reference line must be present. The control line confirms that the test has been performed correctly.

Procedure

The Visitect CD4 LFA test kit includes a disposable test device, a buffer bottle, a 30 µL micropipette, a safety lancet, and an alcohol swab. The testing procedure was conducted in line with the manufacturer's instructions. First, a 30 µL of the sample was taken from the well-mixed 2 mL of whole blood transferred to a 2 mL container for CD4+ T-cell count using micropipette. The blood was dispensed into Well A of the test device and allowed to incubate for 3 minutes. During this time, red blood cells and monocytes were retained in the blood sample collection pad. Next, a drop of buffer was added to Well A and incubated for 17 minutes. This step allowed other white blood cells, including CD4+ T-cells, to migrate to the reaction area where cell lysis occurred, releasing full-length CD4 for capture on the test strip. Following this, three drops of buffer were added to Well B, facilitating the release of the colloidal gold-labelled monoclonal antibody conjugate, which formed the reference line and the control line. The test results were read after 20 minutes by visually comparing the colour intensity of the test line (T) to the reference line (200).

Result Interpretation

A test line darker than the reference line indicated a CD4 cell count greater than 200 cells/mm³, while a test line of similar or fainter intensity suggested a CD4 cell count below 200 cells/mm³ (Figures 3 and 4).

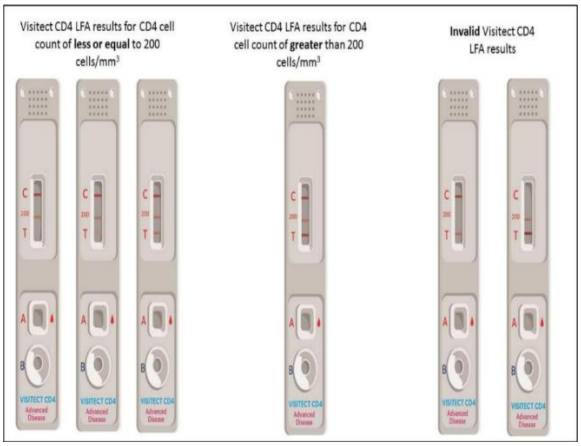

Figure 3: VISITECT® CD4 Advanced Disease Rapid test kit.

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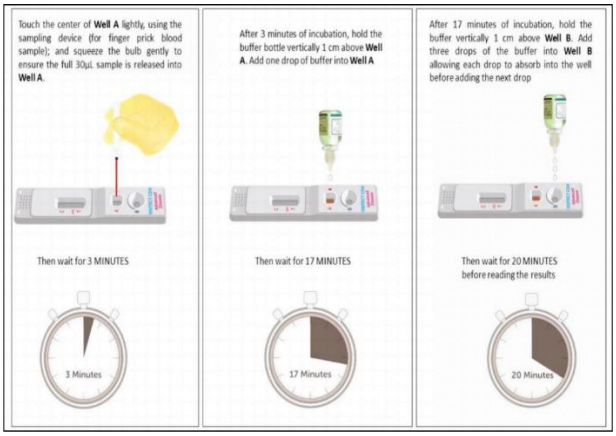

Figure 4: Interpretation of VISITECT® CD4 Advanced Disease Rapid test kit results.

RESULTS

Table 1. Demographic pattern and clinical characteristics of HIV-1 virally Suppressed and Virological Failure Groups in Minna, Niger State

Demographic Information		Virally Suppressed (n=50)		Virological Failure (n=43)	
		Frequency	Percentage (%)	Frequency	Percentage (%)
Age (years)	23 – 32	9	18	2	4.6
	33 – 42	20	40	11	25.6
	43 – 52	17	34	18	41.9
	53 – 62	3	6	10	23.3
	63 – 72	1	2	1	2.3
	73 – 82	0	0	1	2.3
Gender	Male	27	54	32	74.4
	Female	23	46	11	25.6
Educational Status	Formal	34	64	25	58.1
	Informal	16	32	18	41.8
Marital Status	Married	35	70	27	62.7
	Single	8	16	6	13.9
	Divorced	1	2	2	4.7
	Widower	6	12	8	18.6
Occupation	Employed	43	86	25	58.1
	Unemployed	7	14	18	41.9
Family Status	Polygamy	20	40	19	44.1
	Monogamy	30	60	22	51.1
	Unknown	0	0	2	4.6
Transmission Risk	Sexual	47	94	39	90.6
	Transfusion	2	4	2	4.6
	I.V Drugs	1	2	1	2.3
	Unknown	0	0	1	2.3
Adherence Status	Good	33	66	2	4.6
	Fair	15	30	28	65.1
	Poor	2	4	13	30.2
Opportunistic Infection	Bacterial	8	16	8	18.6
	Viral	5	10	13	30.2
	Parasitic	3	6	9	20.9
	Fungi	0	0	0	0
	Multiple	2	4	13	30.2
	Non	32	64		
WHO Staging before cART initiation	1 and 2	47	94	25	58.1
	3 and 4	3	6	18	41.9
CD4 Estimation	≥200	50	100	1	2.3
	<200	0	0	42	97.7

Viral Load @ enrolment	≥1000	50	100	43	100
	<1000	0	0	0	0
Viral Load @ 3-6 months	≥1000	0	0	43	100
	<1000	50	100	0	0

DISCUSSION

Table 1 presents a detailed demographic and clinical profile of the study participants, differentiating between those with virological suppression (VS) and virological failure (VF). The findings indicate notable disparities between the two groups. In the virological failure group, older individuals (43-52 years and 53-62 years) and males (74.42%) were predominant. This contrasts with the virally suppressed group, which showed a more balanced age distribution and gender ratio. This observation aligns with existing literature suggesting that older age can be a risk factor for virological failure, potentially due to age-related immune senescence, comorbidities, or challenges with long-term adherence (Agegnehu et *al.,* 2022; Phiri *et al.,* 2022). The higher proportion of males experiencing virological failure in this study warrants further investigation, as some studies have indicated sex-specific differences in ART adherence or treatment outcomes (Zhou *et al.,* 2022). Regarding educational status, formal education was more prevalent among the virally suppressed (64%) compared to those with virological failure (58.1%). This aligns with the broader understanding that higher educational attainment can correlate with better health literacy, adherence to complex medical regimens, and engagement with healthcare services (Wardhani *et al.,* 2023). Similarly, higher employment rates in the virally suppressed group (86% vs. 58.1% in VF) suggest that socioeconomic stability may positively influence treatment outcomes, potentially by facilitating consistent access to medication and nutritional support. This underscores the importance of addressing socioeconomic determinants of health in HIV care, particularly in resource-limited settings like Minna, Niger State. Sexual transmission remained the primary mode of HIV acquisition in both groups (94% in VS, 90.6% in VF), consistent with the generalized heterosexual epidemic observed in sub-Saharan Africa (Torrone *et al.,* 2018). The most striking difference between the groups was in adherence status and clinical staging. A vast majority of virally suppressed participants reported good adherence (66%), while poor and fair adherence predominated in the virological failure group (30.2% and 65.1%, respectively). This finding strongly reinforces the well-established principle that adherence is the cornerstone of successful ART, as suboptimal adherence directly leads to treatment failure and drug resistance (Anyaike *et al*., 2019; Wensing *et al*., 2023). Furthermore, a significant portion of the virological failure group presented in advanced WHO clinical stages (3 and 4) before cART initiation, and nearly all (97.67%) had CD4 counts below 200 cells/mm³ at enrolment. This indicates severe immunosuppression, which is a known predictor of poorer treatment outcomes and increased risk of opportunistic infections. The persistent high viral loads (≥1000 copies/mL) in the virological failure group at 3-6 months follow-up, compared to undetectable levels in the suppressed group, unequivocally confirms the definition of virological failure as per WHO guidelines (Bogale *et al.,* 2022).

Conclusion

This study demonstrates that HIV-1 genetic diversity in Minna Metropolis, Niger State, is predominantly driven by CRF02_AG, followed by subtype G, A1, and recombinant strains. The high prevalence of resistance-associated mutations (notably M184V/I, K103N, and Y181C) among patients with virological failure underscores the urgent need for continuous surveillance of HIV-1 genetic subtypes and drug resistance patterns in Nigeria. Our findings further confirm the critical role of adherence, socioeconomic stability, and early initiation of ART in determining treatment outcomes. Strengthening viral load monitoring, incorporating routine genotypic resistance testing, and adapting ART regimens to local subtype distributions will be essential to achieving Nigeria's 95-95-95 HIV targets. Targeted health education, adherence counseling, and social support interventions should also be prioritized, particularly for older patients, males, and individuals with low socioeconomic stability. Overall, molecular epidemiology remains central to guiding effective HIV control strategies in the region.

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