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Virologic non-suppression determinants in Kenyan HIV positives

A cross-sectional study

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Abstract

Despite the substantial scale-up in treatment coverage and viral load monitoring, virologic non-suppression persists among people living with HIV in Kenya. In this study, we describe the extent of virologic non-suppression and identify associated factors in people living with HIV under combination antiretroviral therapy and routine viral load monitoring in Kenya. We conducted a retrospective cross-sectional study on routinely collected viral load data generated from January 2016 to June 2025. Mean, median, and proportions were used to describe the variables, and logistic regression analysis was performed to determine the factors associated with virologic non-suppression. The overall virologic non-suppression rate was 7.1%. Increased odds of virologic non-suppression were associated with male sex (adjusted odds ratio [aOR] = 1.35, 95% confidence interval [CI]: 1.33–1.37, $P < .001$), children (aOR = 2.19, 95% CI: 2.14–2.24, $P < .001$), adolescents (aOR = 1.77, 95% CI: 1.73–1.81, $P < .001$), Protease inhibitor based regimen (aOR = 1.12, 95% CI: 1.10–1.15, $P < .001$), and persistent low-level viremia (aOR = 1.46, 95% CI: 1.24–1.70, $P < .001$). Longer duration on treatment (73+: aOR = 0.95, 95% CI: 0.93–0.97, $P < .001$) and Integrase inhibitor-based regimen (aOR = 0.29, 95% CI: 0.28–0.29, $P < .001$) were protective of non-suppression. Virologic non-suppression was common in the study population. Sex, age, duration on treatment, regimen class, and geographical location were significantly associated with non-suppression. Each of these variables presents opportunities for interventions to help achieve the UNAIDS 95-95-95 goals.

Abbreviations: 3TC = lamivudine, ABC = abacavir, AIDS = acquired immunodeficiency syndrome, aOR = adjusted odds ratio, ART = antiretroviral therapy, ASAL = arid and semi-arid land, BPI = boosted protease inhibitor, cART = combination antiretroviral therapy, CCC = comprehensive care clinic, DTG = dolutegravir, EFV = efavirenz, HIV = human immunodeficiency virus, INSTI = integrase strand transfer inhibitor, LPVr = lopinavir, NASCOP = National AIDS and STI Control Programme, NNRTI = non-nucleoside reverse transcriptase inhibitor, NRTI = nucleoside reverse transcriptase inhibitor, NVP = nevirapine, PI = protease inhibitor, PLHIV = people living with human immunodeficiency virus, TDF = tenofovir disoproxil fumarate, VL = viral load, VLNS = virologic non-suppression, VLS = virologic suppression.

Keywords: antiretroviral therapy (ART), HIV/AIDS, non-suppression (VLNS), suppression (VLS), viral load (VL)

1. Introduction

Human immunodeficiency virus (HIV) continues to be a significant public health problem despite enormous efforts to manage it. Globally, there were 40.8 million people living with HIV (PLHIV) in 2024, 31.6 million of whom had access to antiretroviral therapy (ART).[1] As of 2024, Kenya had an estimated 1.33 million people living with HIV, of whom approximately 90% were receiving antiretroviral therapy. Of these, 95% had achieved virologic suppression, a notable decrease from 97% in 2023.[2] Despite the substantial scale-up in treatment coverage and viral load (VL) monitoring, virologic non-suppression (VLNS) persists among people living with HIV in Kenya.[3-7]

The decline in suppression levels in the HIV positive population in Kenya necessitates the evaluation of potential key predictors of virologic non-suppression since it has been linked to an increased risk of progression to clinical failure, a higher risk of viral transmission, and the emergence of HIV drug resistant

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The data that support the findings of this study are available from a third party, but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are available from the authors upon reasonable request and with permission of the third party.

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mutations.[8,9] Adequate monitoring of viral load levels is essential in monitoring treatment effectiveness, identifying patients needing enhanced adherence counseling, and in the early detection of treatment failure. Virologic non-suppression in the HIV positive population in Kenya has been associated with long periods on antiretrovirals (ARVs), certain ARV regimens, advanced HIV, low level viremia, sex, age, and drug resistance.[5,6,10-12] Thus, monitoring virologic non-suppression and viral rebound, and evaluating the associated factors can facilitate the design of effective interventions.[3] Leveraging routinely collected programmatic data is imperative in the development and implementation of evidence-based policies and guidelines in HIV care and management in Kenya.

We aimed to describe the extent of virologic non-suppression and identify associated factors in people living with HIV under antiretroviral therapy and routine VL monitoring between 2016 and 2025 in Kenya.

2. Materials and methods

2.1. Study design and population

We conducted a retrospective cross-sectional study on routinely collected VL data generated from January 2016 to June 2025. Records of HIV-positive patients enrolled at Comprehensive Care Centers (CCCs) under the National AIDS and STI control program (NASCOP) VL national program were analyzed. Our study included all PLHIV of all age groups and all genders on combination antiretroviral therapy (cART) under VL monitoring.

2.2. Data source

Data analyzed in this study was extracted from the NASCOP database. Patient identifiers such as names were removed while retaining the unique patient CCC numbers. Abstracted variables included demographics (age, sex, date of birth, CCC name, facility code, county, sub-county, supporting partner) and clinical data (sample type, reason for VL test, ART initiation date, current regimen, dates of sample collection/testing/result dispatch, and viral load in copies/mL).

2.3. Ethical considerations

Approval for the study was sought and received from the Scientific Ethical Review Unit (SERU) of the Kenya Medical Research Institute: Protocol No. KEMRI/SERU/CIPDCR/031/3707. This study was conducted in accordance with the principles established by the 18th World Medical Assembly General Assembly (Helsinki, 1964) and in full conformance with ICH Good Clinical Practices guidelines and Good Laboratory Practices guidelines. Since this study accessed secondary data with no participant identifiers, consent was waived.

2.4. Outcome and predictors of VLNS

The main outcome of this study was binary: VLNS and VLS. VLNS was defined as a single VL of ≥1000 copies/mL after at least 6 months of cART. We examined gender (male, female), age in years (0–14, 15–24, 25–44, 45+), treatment duration in months (0–6, 7–24, 25–48, 49–72, 73+), drug class (integrase strand transfer inhibitor [INSTI]-based, protease inhibitor [PI]-based, non-nucleoside reverse transcriptase inhibitor [NNRTI]-based, third line, other), justification (routine VL, baseline, clinical failure, persistent low level viremia [PLLV], prevention of mother-to-child transmission [PMTCT], recency, single drug substitution), and geolocation (arid and semi-arid land [ASAL], non-ASAL). The following sub-categories were set as the reference levels: female for gender, 25 to 44 for age group, 49 to 72 for treatment duration, NNRTI-based for drug class, Routine VL for justification, and non-ASAL for geolocation.

2.5. Statistical analysis

We used medians with interquartile ranges (IQR) and counts with proportions to describe continuous and categorical variables, respectively. Observations with missing and discrepant data were excluded from the analysis. The Wilcoxon rank sum test was used to compare categorical variables, while Pearson Chi-squared test was used to compare continuous variables. A binomial logistic regression model was fit to the data using R version 4.4.3, with the stats package.[13] All statistical tests were 2-sided, and the significance level was set at 5% ($P = .05$).

3. Results

3.1. Baseline characteristics of the study population

We included 1,160,722 PLHIV in our study (Fig. 1), 7.1% of whom were virologically non-suppressed. Table 1 shows the sociodemographic and clinical characteristics by virologic suppression status based on the most recent VL measurement. Younger PLHIV and those with ≤ 6 months exhibited higher rates of virologic non-suppression.

The overall median age at VL testing was 39 years and the median duration on ART was 60 months. The median age of non-suppressed patients (33 years) was lower than that of suppressed patients (39 years). The median treatment duration was also lower in non-suppressed patients than in suppressed patients (Table 1).

Twenty-five distinct cART regimens were available and in use, each comprising a backbone of 2 Nucleoside Reverse Transcriptase Inhibitors (NRTIs) combined with a third, fourth, or fifth agent classified as either an NNRTI, an INSTI, or a PI. Tenofovir disoproxil fumarate combined with lamivudine and dolutegravir (TDF + 3TC + DTG) and Tenofovir disoproxil fumarate combined with lamivudine and efavirenz (TDF + 3TC + EFV) were the most common regimens in use, accounting for 55.7% and 25% of all regimens. ABC + 3TC + NVP (28.6%), ABC + 3TC + LPVr (26.5%), and ABC + 3TC + EFV (22.9%) exhibited the highest levels of non-suppression. By drug class, protease inhibitor-based regimens showed the highest overall non-suppression (13.4%), while integrase inhibitor-based regimens showed the lowest (3.3%; Table 1).

3.2. Unadjusted regression analysis

Children aged 0 to 14 years and adolescents aged 15 to 24 years had significantly higher odds of VLNS than young adults aged 25 to 44 years (OR = 3.02 (2.96–3.09) and OR = 2.02 (1.98–2.06) respectively). Male PLHIV also had increased odds of non-suppression compared to females (OR = 1.20 (1.18–1.22)). Relative to NNRTI-based regimens, protease inhibitor-based regimens were associated with increased odds of VLNS (OR = 1.45 (1.42–1.48)), whereas INSTI-based regimens were associated with substantially lower odds of virologic non-suppression (OR = 0.27 (0.26–0.27)). Long treatment duration of more than 73 months also exhibited reduced odds of non-suppression (OR = 0.88 (0.86–0.907); Table 2).

Twelve of the twenty-five cART regimens, especially those containing PIs and NNRTIs, were significantly associated with an elevated risk of virologic non-suppression. ABC + 3TC + NVP (OR = 3.51 (3.25–3.79)) and ABC + 3TC + LPVr (OR = 3.22 (3.09–3.34)) had the highest risk of failure, exhibiting more than 3-fold odds of non-suppression than TDF + 3TC + EFV. In contrast, dolutegravir-containing cART regimens were significantly associated with reduced odds of non-suppression (Fig. 2).

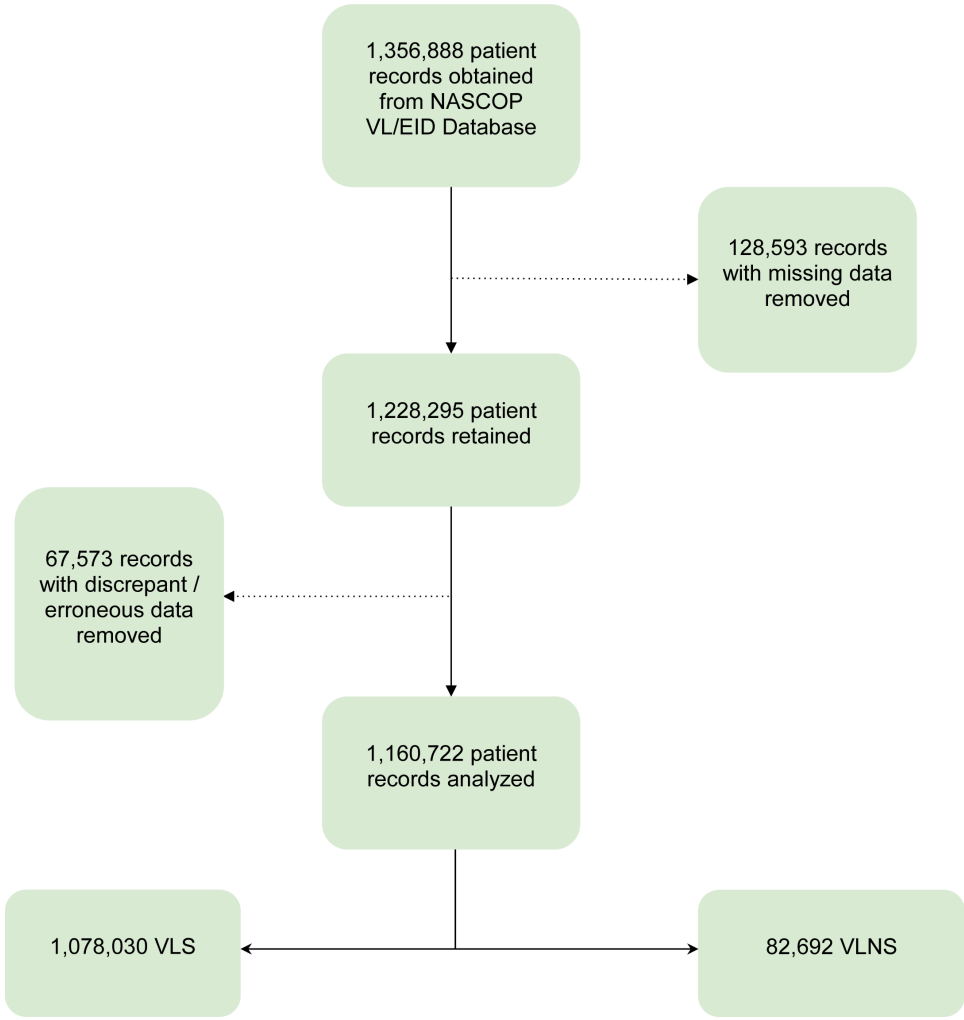


Figure 1. Inclusion and exclusion criteria for the VL dataset observations. NASCOP = National AIDS and STI Control Programme, VL = viral load, VLNS = virologic non-suppression, VLS = virologic suppression.

There were significant geographic disparities in the risk of virologic non-suppression. ASAL regions were significantly associated with an increased risk of non-suppression compared to Nairobi County, with Mandera, Samburu, and Marsabit exhibiting the highest odds (Fig. 3).

3.3. Adjusted regression analysis

Multivariable logistic regression revealed that sex, age, treatment duration, cART regimen class, and geographical location were significantly associated with virologic non-suppression. Adjusting for other variables revealed that male PLHIV had a 35% higher risk of VLNS than female PLHIV (adjusted odds ratio [aOR] = 1.35 (1.33–1.37)). Children and adolescents were almost 2 times more likely to experience non-suppression than young adults (aOR = 2.19 (2.14–2.24) and aOR = 1.77 (1.73–1.81) respectively). Increased odds of virologic non-suppression were also associated with a shorter treatment duration of less than forty-eight months, as depicted in 0–6 months (aOR = 2.15 (2.07–2.23)) and 7–24 months (aOR = 1.42 (1.39–1.45)).

Although PI-based regimens were still associated with increased odds of VLNS after adjusting for other variables, the effect size reduced significantly (aOR = 1.12 (1.10–1.15)). Notably, persistent low-level viremia and single drug substitution showed a significant association with non-suppression (aOR = 1.46 (1.24–1.70) and aOR = 0.72 (0.67–0.76), respectively; Table 2).

4. Discussion

We examined the levels of virologic non-suppression in PLHIV on antiretroviral therapy between 2016 and 2025 and found that 7.1% of our study population was virologically non-suppressed at the most recent VL test. These suppression rates were lower than the UNAIDS target of 95% and have only marginally improved with the widespread use of dolutegravir. It is plausible that drug resistance-conferring mutations contribute significantly to virologic non-suppression in our population, with early signs of emerging resistance to dolutegravir.

We observed that 69% of the study population were female PLHIV. In Kenya, the ratio of females to males living with HIV is 63.62% to 36.38%, implying that there is a slight excess of observations attributable to females. This bias is likely due to the impact of PMTCT programs and better health-seeking behavior and adherence among female PLHIV. Notably, the odds of virologic non-suppression were also elevated in male PLHIV. The increased risk of treatment failure in men has previously been linked to poor health-seeking behaviors such as refusal to seek treatment,[14] presenting later for care with more advanced stages of HIV disease,[15-17] and defaulting treatment.[18,19] Suggestions have been made to design targeted interventions such as setting up men-friendly environments: using male healthcare workers, men-only facilities, or men-only hours for additional adherence sessions.[18,20]

Table 1

Demographic and clinical characteristics of the study participants.

Characteristic	VLNS N = 82,692*	VLS N = 1,078,030*
Age (yr)	33 (20, 43)	39 (31, 48)
Treatment duration (mo)	47 (19, 85)	61 (29, 98)
Gender		
Female	53,736 (65%)	743,680 (69%)
Male	28,956 (35%)	334,350 (31%)
Age category		
0–14	14,103 (17%)	66,732 (6.2%)
15–24	12,433 (15%)	88,092 (8.2%)
25–44	39,347 (48%)	562,813 (52%)
45+	16,809 (20%)	360,393 (33%)
Treatment duration category		
0–6	4990 (6.0%)	32,140 (3.0%)
7–24	19,860 (24%)	190,806 (18%)
25–48	16,942 (20%)	208,336 (19%)
49–72	13,690 (17%)	197,994 (18%)
73+	27,210 (33%)	448,754 (42%)
Justification		
Routine VL	67,724 (82%)	1,009,526 (94%)
Baseline	910 (1.1%)	2679 (0.2%)
Clinical failure	147 (0.2%)	629 (<0.1%)
No data	56 (<0.1%)	780 (<0.1%)
Other	94 (0.1%)	506 (<0.1%)
PLLV	179 (0.2%)	2143 (0.2%)
PMTCT	95 (0.1%)	895 (<0.1%)
Recency	54 (<0.1%)	40 (<0.1%)
Repeat VL	12,413 (15%)	31,859 (3.0%)
Single drug substitution	1020 (1.2%)	28,973 (2.7%)
Regimen class		
INSTI-based	22,210 (27%)	644,180 (60%)
NNRTI-based	43,793 (53%)	336,914 (31%)
PI-based	14,954 (18%)	79,457 (7.4%)
Third-line	26 (<0.1%)	189 (<0.1%)
Other	1709 (2.1%)	17,290 (1.6%)
County geolocation		
Non-ASAL	63,541 (77%)	897,571 (83%)
ASAL	19,151 (23%)	180,459 (17%)

ASAL = arid and semi-arid land, INSTI = integrase strand transfer inhibitor, NNRTI = non-nucleoside reverse transcriptase inhibitor, PI = protease inhibitor, PLLV = persistent low-level viremia, PMTCT = prevention of mother-to-child transmission, VL = viral load, VLNS = virologic non-suppression, VLS = virologic suppression.

*Median (IQR) or frequency (%).

The increased odds of virologic non-suppression depicted by younger age groups in our study are consistent with previous studies that have reported that older PLHIV are more likely to achieve virologic suppression.[21-23] Several factors may contribute to non-suppression in these age groups, including poor ART adherence,[24-26] substance abuse,[25] mental distress, and non-disclosure of their HIV status to their loved ones.[27,28] Research studies have also reported HIV-related stigma as a significant factor affecting drug adherence and treatment outcomes in younger patients.[29,30] Studies show that alcohol and recreational drug use and low socioeconomic status contribute to non-adherence, thus consequently resulting in virologic failure in younger patients.[31,32] Constant monitoring by peer supporters, social workers, or healthcare workers could help younger adults manage stigma and other issues hampering their treatment, hence improving ART adherence and achieving viral suppression.

We showed that NNRTI-based and PI-based regimens were associated with increased odds of virologic non-suppression, whereas integrase inhibitor-based regimens were protective. NNRTIs have a low genetic barrier; thus, a single mutation can result in high-level resistance. Clinical cross-resistance has also been reported in NNRTIs where the presence of NNRTI mutations results in low susceptibility to multiple NNRTIs. A survey done by WHO in 2022 reported that the levels of pretreatment drug resistance (PDR) to nevirapine and efavirenz in populations initiating first-line ART were above 10%, and treatment-experienced patients were 3 times more likely to develop PDR to NNRTIs.[33] Drug resistance mutations are known to accumulate steadily over the course of treatment, which may explain the relative ineffectiveness of low genetic-barrier regimens still in use.[34,35]

Resistance to boosted PIs is difficult to evolve, and, in the presence of boosted protease inhibitors (bPIs), it is unlikely that resistance evolves to the other drugs the patient is taking. This protective effect of bPIs can be explained, in part, by the low plasma viral loads achieved using bPIs. There has also been evidence to suggest that resistance to NRTIs is unlikely to emerge even in patients with viremia who are on boosted PIs via a yet-to-be-described mechanism.[36] Despite this, our data suggests that the situation is now markedly different, and PIs, whether boosted or not, face increased odds of VLNS. Poor adherence and drug resistance are plausible explanations for this ineffectiveness, although our data does not indicate which, if either, is the primary driver; further research will be required to determine the proximate cause.

The WHO Global action plan on HIV drug resistance 2017 to 2021 emphasized the transition to DTG-containing regimens since they've been shown to completely suppress viral loads to undetectable levels.[37] In our environment, dolutegravir-containing regimens have continued to be effective, likely due to better adherence, low pill burden, tolerability, and its high genetic barrier, since multiple resistance mutations must accumulate before it loses efficacy.[38] In Sub-Saharan Africa, a low prevalence of major INSTI resistance mutations occurring at a low-level detection threshold has been reported, hence affirming the potential effectiveness of INSTI-based regimens and the reduced risk of non-suppression revealed in our study population.[39]

Despite the impressive ART and VL scale-up in Kenya, geographical disparities persist. Our data showed that ASAL regions had the highest prevalence of virologic non-suppression even though they had fewer people living with HIV than other regions. These regions were also significantly associated with increased odds of virologic non-suppression. For instance, patients living in Samburu were approximately 4 times more likely to experience VLNS when compared to those living in Nairobi. Samburu and other ASALs such as Mandera, Marsabit, and Wajir are characterized by inaccessibility due to poor road networks, nomadic pastoralism, and terrorist activities. This results in delays in the delivery of essential healthcare resources such as drugs, resulting in drug stock-outs. Treatment interruptions and intermittent uptake of ART have been linked to viral load relapse[9,40,41] and to a greater risk of development of HIV drug resistance.[42] Insecurity in such regions has also stalled the setting up, development, and implementation of healthcare resources and programs targeted toward HIV-positive patients.

Proper treatment adherence has been shown to dramatically reduce viral load below detectable levels by the most sensitive assays of up to <50 copies/mL. The WHO 2015 guidelines indicate that virologic suppression can be achieved after at least 12 months of adherence. In our study, a treatment duration of more than 73 months was associated with a 3% reduced risk of VLNS. This negative association could be due to sustained adherence which reduces viral replication resulting in virologic suppression. On the other hand, long treatment duration has also been previously associated with virologic failure.[10,43] Increased risk of treatment failure has been linked to increased lack of drug tolerability, accumulation of HIV drug resistance mutations, development of drug side effects, and the emergence of multi-strain viruses.[44,45]

4.1. Study limitations

A notable limitation of this study was the absence of medication adherence data. The available dataset lacked critical adherence indicators such as on-time pill pick-up and consistent

Table 2

Association of sociodemographic and clinical variables with VLNS.

	Univariable analysis			Multivariable analysis		
Characteristic	**OR**	**95% CI**	***P*-value**	**aOR**	**95% CI**	***P*-value**
Gender						
Female	–	–		–	–	
Male	1.20	1.18, 1.22	<.001	1.35	1.33, 1.37	<.001
Age category						
25–44	–	–		–	–	
0–14	3.02	2.96, 3.09	<.001	2.19	2.14, 2.24	<.001
15–24	2.02	1.98, 2.06	<.001	1.77	1.73, 1.81	<.001
45+	0.67	0.65, 0.68	<.001	0.81	0.79, 0.82	<.001
Treatment duration category						
49–72	–	–		–	–	
0–6	2.25	2.17, 2.32	<.001	2.15	2.07, 2.23	<.001
7–24	1.51	1.47, 1.54	<.001	1.42	1.39, 1.45	<.001
25–48	1.18	1.15, 1.20	<.001	1.09	1.07, 1.12	<.001
73+	0.88	0.86, 0.90	<.001	0.95	0.93, 0.97	<.001
Justification						
Routine VL	–	–		–	–	
Baseline	5.06	4.69, 5.46	<.001	2.62	2.41, 2.86	<.001
Clinical failure	3.48	2.90, 4.16	<.001	3.32	2.74, 4.00	<.001
No data	1.07	0.81, 1.39	.6	1.31	0.98, 1.71	.060
Other	2.77	2.21, 3.43	<.001	1.62	1.29, 2.03	<.001
PLLV	1.25	1.07, 1.45	.005	1.46	1.24, 1.70	<.001
PMTCT	1.58	1.27, 1.94	<.001	1.21	0.97, 1.49	.086
Recency	20.1	13.4, 30.5	<.001	20.5	13.5, 31.4	<.001
Repeat VL	5.81	5.68, 5.94	<.001	4.56	4.45, 4.67	<.001
Single drug substitution	0.52	0.49, 0.56	<.001	0.72	0.67, 0.76	<.001
Regimen class						
NNRTI-based	–	–		–	–	
INSTI-based	0.27	0.26, 0.27	<.001	0.29	0.28, 0.29	<.001
PI-based	1.45	1.42, 1.48	<.001	1.12	1.10, 1.15	<.001
Third-line	1.06	0.69, 1.56	.8	0.76	0.48, 1.15	.2
Other	0.76	0.72, 0.80	<.001	0.52	0.49, 0.55	<.001
County geolocation						
Non-ASAL	–	–		–	–	
ASAL	1.50	1.47, 1.52	<.001	1.66	1.63, 1.69	<.001

aOR = adjusted odds ratio, ASAL = arid and semi-arid land, CI = confidence Interval, INSTI = integrase strand transfer inhibitors, NNRTI = non-nucleoside reverse transcriptase inhibitor, OR = odds ratio, PI = protease inhibitor, PLLV = persistent low-level viremia, PMTCT = prevention of mother-to-child transmission, VL = viral load, VLNS = virologic non-suppression, VLS = virologic suppression.

clinic attendance, which are essential for accurately assessing treatment adherence. The retrospective design constrained the analysis to existing records within the national viral load (VL) database. Incorporating these adherence-related variables into logistic regression models is crucial for a comprehensive evaluation of their contribution to the risk of treatment failure.

The national HIV viral load and early infant diagnosis programs in Kenya do not routinely incorporate drug resistance testing or plasma antiretroviral level monitoring. Consequently, genotyping of the virus prior to treatment initiation to identify resistance-associated mutations is not standard clinical practice. Evidence indicates that performing baseline genotypic resistance testing and selecting a fully active antiretroviral drug combination significantly improves treatment outcomes among patients with transmitted drug resistance. In the context of our study, incorporation of genotyping and plasma drug level measurements would have provided critical insights into the underlying causes of virologic non-suppression in a substantial subset of patients. Additionally, such analyses could have aided in differentiating virological failure due to resistance from that resulting from poor adherence.

5. Conclusion

Levels of VLNS have declined progressively over recent years; however, the current rate of decline remains inadequate for Kenya to meet the UNAIDS 95-95-95 targets by 2030. Male patients, individuals receiving NNRTI and PI-based regimens, as well as younger patients, exhibit a higher risk of experiencing VLNS. The poor efficacy of several antiretroviral regimens necessitates the expedited transition to the more efficacious and less resistance-prone Dolutegravir-based regimens. It is therefore critical to establish and sustain targeted follow-up programs aimed at ensuring treatment adherence and minimizing loss to follow-up. Furthermore, routine drug resistance testing for both virologically suppressed and non-suppressed individuals is essential to guide and optimize personalized antiretroviral therapy regimens. Considerable disparities in VLNS prevalence at the county level warrant the development of a risk assessment tool to facilitate the equitable allocation of resources related to HIV care and management.

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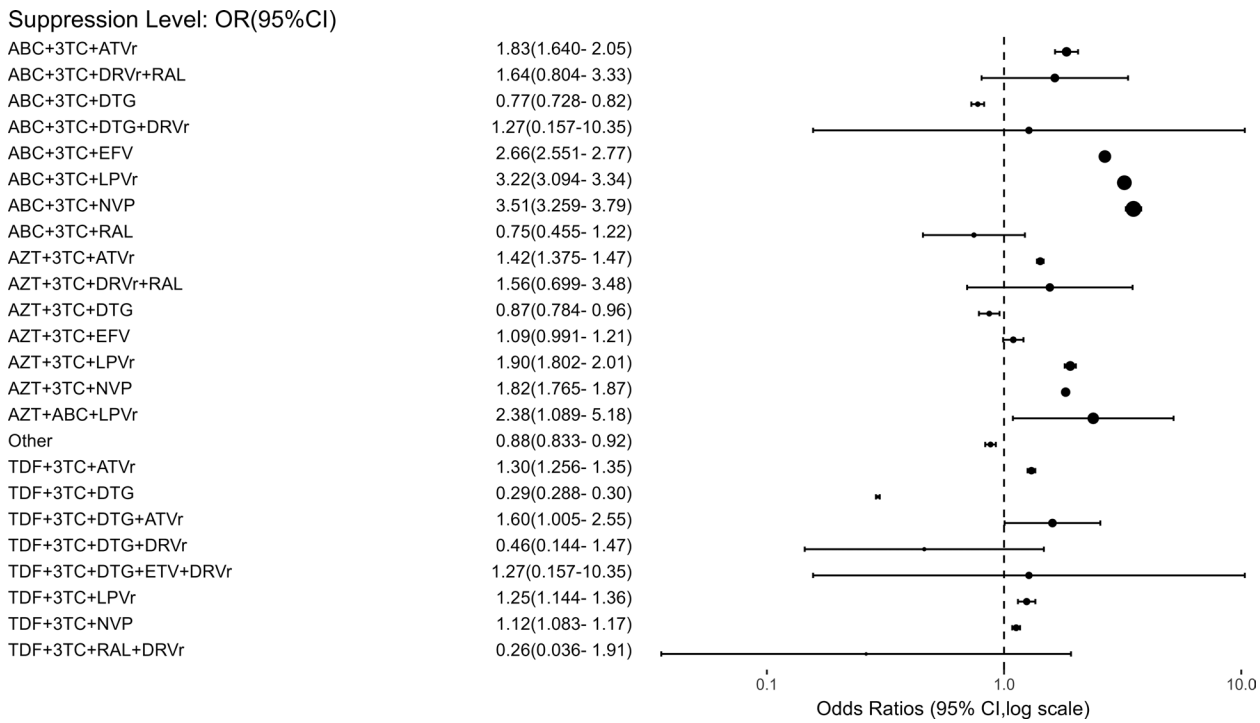


Figure 2. Forest plot showing the association between virologic non-suppression and ART regimen administered. TDF + 3TC + EFV was set as the reference category (OR = 1). 3TC = lamivudine, ABC = abacavir, ART = antiretroviral therapy, CI = confidence interval, DTG = dolutegravir, EFV = efavirenz, LPVr = lopinavir, NVP = nevirapine, OR = odds ratio, TDF = tenofovir disoproxil fumarate.

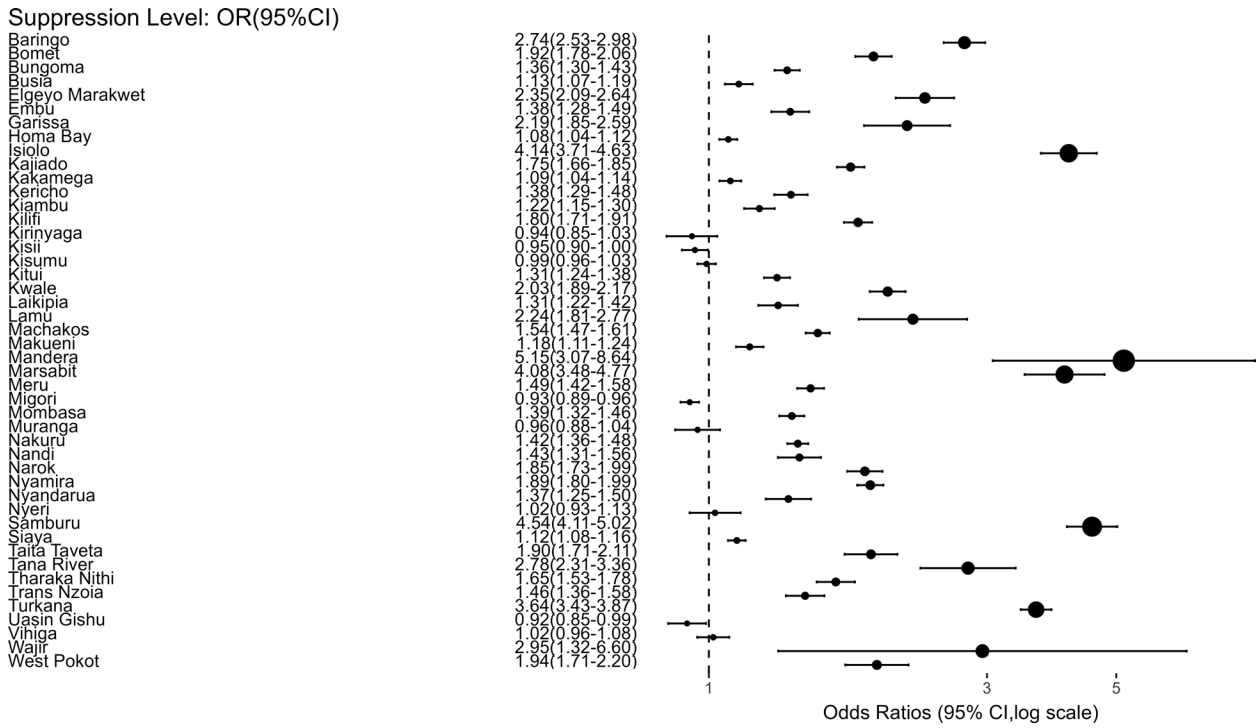


Figure 3. Forest plot showing the association between virologic non-suppression and counties. Nairobi was set as the reference category (OR = 1). CI = confidence interval, OR = odds ratio.

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