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Three-year outcomes after genotyping-guided treatment switch in HIV patients with virologic failure in Cameroon: Implications for epidemic control in low- and middle-income countries

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ABSTRACT

Objectives: Monitoring of HIV-infected patients with long-term treatment remains challenging in low- and middle-income countries (LMICs) due to risks of multi-class HIV drug resistance (HIVDR). We sought to evaluate the treatment response after a genotypic resistance-guided treatment switch among patients with multi-class HIVDR in Cameroon.

Methods: A retrospective cohort study was conducted in the South-West region of Cameroon among patients failing first- and second-line antiretroviral treatment (ART) (i.e., non-nucleoside reverse transcriptase inhibitors (NNRTI)- and protease inhibitor (PI)-based) from 2018 to 2023. Following HIV-1 genotypic resistance testing (GRT), patients were switched to the most effective genotype-guided therapies and viral load (VL) was monitored thereafter. Data analysis was performed using Epi-Info v.7.2, with $P < 0.05$ considered significant.

Results: Overall, 81 patients were included in the study (median CD4 and VL were 232.5 [161.25–401.25] cells/µL and 54,480 [13,932.5–220,153] copies/mL, respectively). HIVDR at failure (i.e., VL>1000copies/mL) was 61.7%, and prevailing mutations were M184V (24.6%), K103N (17%) and M46I (32.1%) for NRTI, NNRTI and PI respectively. Monitoring data revealed 92.6% of adherence to the prescribed regimen. Viral suppression post-GRT was 80% at 3-months, 84.8% at 6-months, 81.4% at 12-months, 93.7% at 24-months

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and 86.9% at 36-months. Furthermore, DTG-based regimens yielded significantly higher responses than DTG-sparing regimens up to 24-months ($P = 0.013$, $P = 0.0015$, $P = 0.0005$, $P = 0.02$, respectively).
Conclusions: Our findings highlight sustained viral suppression over three years following GRT-guided switch among individuals with multi-class HIVDR, mainly driven by DTG-based regimens. This underscores the significance of personalizing ART-management for difficult-to-treat people to achieve HIV control by 2030 in LMICs.

1. Introduction

HIV/AIDS-related morbidity and mortality have significantly decreased in the past two decades, thanks to diagnosis, treatment, and monitoring programs worldwide [1–3]. Yet as antiretroviral therapy (ART) has become lifelong and exposure durations have lengthened, HIV drug resistance (HIVDR) has emerged as a major threat to sustained viral suppression, particularly among people with long treatment histories who accumulate multidrug-resistant (MDR) variants and face limited treatment options [4,5].

In sub-Saharan Africa (SSA), pretreatment and acquired resistance to non-nucleotide reverse transcriptase inhibitors (NNRTIs) are high, with increasing trends of HIVDR documented across other classes, substantially raising the risk of virologic failure and onward transmission; thus underscoring the need for routine drug resistance surveillance and responsive treatment strategies [6,7]. Despite global progress, many SSA countries remain off-track for the 'third 95′ (viral load suppression) target. In 2024, an estimated 74% of adults living with HIV globally had suppressed viral loads; Western and Central Africa (including Cameroon) continue to lag behind Eastern and Southern Africa, reflecting persistent gaps along the cascade [8]. These shortfalls are driven by system-level impediments like limited access to routine viral load testing and genotypic resistance testing (GRT), delayed treatment switch and biological barriers such as widespread HIVDR [8].

Cameroon, like many SSA countries, uses a public-health approach to antiretroviral therapy (ART), switching patients to preestablished second-line regimens (protease inhibitor (PI)-based) after their first-line ART (NNRTI-based) has failed, instead of considering their specific patterns of antiretroviral (ARV) resistance. In the presence of cross-resistance between first- and second-line drugs, this strategy can place patients on suboptimal therapy and perpetuate viremia [9]. By contrast, genotypic drug-resistance testing (GRT) determines which HIV medications, if any, are still effective against an individual's HIV and enables rational, resistance-informed regimen selection [10]. When treatment is modified based on resistance test results, salvage regimens have a greater efficacy with earlier virological response compared with empirical changes alone [11–15]. In addition to enhancing the outcomes of individual patients, optimizing treatment for patients with drug-resistance mutations (DRMs) may also contribute to the prevention of the spread of drug-resistant strains and the preservation of the efficacy of the current ART regimen [16]. However, the routine use of GRT for personalized patient management remains limited in many low- and middle-income countries (LMICs) due to cost, laboratory capacity, and turnaround time constraints. Consequently, GRT is more often deployed for surveillance than for clinical decision-making [17–19], unlike in high-income countries, where GRT is used to guide treatment, whether at initiation or before a treatment switch. Given the accumulation of DRMs in long-term patients and programmatic constraints that impede optimal switching, post-GRT-tailored therapy is needed both to improve individual outcomes and to curb the spread of resistant strains. Our study, therefore, targeted patients who underwent GRT after a therapeutic failure, and their regimen was switched following GRT recommendations. We sought to ascertain the virological response of patients failing ART after implementing a resistance-guided regimen optimization strategy.

2. Materials and methods

2.1. Study design and target population

We conducted a retrospective cohort study from 2018 to 2023 in individuals from all age groups who had failed their treatment at three centers in the South-West region and were referred for GRT at the Chantal Biya International Reference Centre for Research on HIV/AIDS prevention and management (CIRCB), Yaoundé, Cameroon.

2.2. Study site description

Our study was conducted at CIRCB in Yaoundé, Cameroon, and at the three centers selected for the study: one rural site (Mutengene Baptist Hospital) and two urban sites (Buea Regional Hospital and Limbe Regional Hospital), all located in the South West Region of Cameroon. With extensive experience in HIV/AIDS research, CIRCB was established in 2006 and is currently the only WHO-accredited center for HIV drug resistance surveillance at the national level [20], providing GRT for PLWHIV at a reduced cost. On the other side, all three clinical sites where chosen based on their important active files and because they were the main centers referring patients for GRT in the region.

2.3. Sampling method

Medical records of eligible participants at the three health centers were consecutively enrolled into the study. Were considered for inclusion all participants on any ART regimen who had been treated for at least 6 months from the selected health centers and who had two consecutive plasma viral loads ≥1000 copies/mL after receiving intensified adherence counselling as per our national guidelines. All participants had undergone genotypic resistance testing (GRT) at CIRCB and had at least two postswitch viral loads (VL) at various time points. We excluded individuals with no postswitch follow-up data (i.e., no VL testing after GRT).

2.4. Laboratory analysis

Briefly, in-house protocol for GRT is as follows [21]; first, 10 mL of blood was collected from treatment-failing patients who presented for GRT at CIRCB. Plasma samples were then separated from whole blood and stored at temperatures between −20 °C and −80 °C when they were not to be analyzed immediately. Viral RNA was extracted using a standard method after spinning 1 mL of plasma at 14,000 rpm to concentrate viral RNA and thus increase PCR sensitivity. Following this step, 140 µL of plasma was retained for HIV-1 RNA extraction following the QIAGEN protocol (QIAamp® DNA

Minikit; QIAGEN, Courtaboeuf, France). Using an in-house protocol and a two-step polymerase chain reaction (PCR), HIV-1 polgene (protease and reverse-transcriptase regions) was then amplified (~1600 base pairs). Using agarose gel electrophoresis, the effectiveness and integrity of PCR products was confirmed, following which the amplicons were purified. Purified products were submitted to a sequencing reaction. The products of the sequencing reaction were then gel-purified using Sephadex (Separation Pharmacia Dextran; Cytiva Sweden AB, SE-751 84 Uppsala, Sweden) gel to remove excess reactants and render the DNA products pure. Sequencing was performed using an Applied Biosystems 3500 genetic analyzer (HITACHI, Tokyo, Japan), and sequence assembly and editing were performed with SeqScape v2.7 (Applied Biosystems) or Recall v2.28 [22]. The Stanford University database (Stanford HIVdb) was used to identify DRMs.

2.5. Sequence interpretation and analysis

All edited sequences were interpreted using Stanford HIVdb v.9.4. to identify major DRMs and potentially active medications. Rapid subtyping tools, such as COMET (https://comet.lih.lu/, accessed on 21 June 2020) and REGA (http://dbpartners.stanford.edu:8080/RegaSubtyping/stanford-hiv/typingtool/, accessed on 21 June 2020), were used for subtyping, with confirmations performed using molecular phylogeny after sequence alignment with BioEdit v.7.2.5 and tree inference with MEGA software v.70.107.

2.6. Data interpretation and statistical analysis

The viral load testing was performed after GRT-guided switch using a commercial kit from Abbott (GmbH & Co. KG, n.d.) [23] at different time intervals. Data generated were then entered into Microsoft excel and analyzed using Epi info v7.2.3.1. Quantitative variables were described using mean (standard deviation) and median (interquartile range). Qualitative variables were described with frequencies and proportions. Associations between qualitative variables were calculated using Fischer's exact test, and *P*-values < 0.05 were considered statistically significant.

2.7. Ethical considerations

Ethical clearance was sought and obtained from the Institutional Review Board of the Faculty of Health Sciences, University of Buea (reference number: 2024/2376–01/UB/SG/IRB/FHS). Research authorization was also obtained from the CIRCB directorate to conduct our study in this establishment and the various health centers. Confidentiality was ensured by using patient identification numbers with password-protected computers, and core ethical values were respected.

3. Results

We identified a total of 336 patients in the South West region and included a total of 81 patients. Fig. 1 shows the complete procedure for enrollment, inclusion, and exclusion of the study.

3.1. Socio-demographic and baseline data

We retained a total of 81 participants, with a mean age (±SD) of 43 (±5.6) years; most were female (58%, n = 47). All three health centers (two urban and one rural) of the SW region were represented, including Buea (n = 21, 26%); Limbe (n = 42, 51.8%); and Mutengene (n = 18, 22.2%) centers. Of the 81 participants, 28 (34.6%) were failing first-line ART with 20 (24.7%) failing specifically non-nucleoside reverse transcriptase inhibitor (NNRTI)-based and 8 (9.9%) failing a dolutegravir (DTG)-based first-line ART (following transition to tenofovir/lamivudine/dolutegravir after prior exposure to efavirenz-based). Notably, about 53 (65.4%) were failing protease inhibitor (PI/r)-based second-line (NB: The second-line failing patients had previously failed NNRTI-based first-line regimens as recommended by national treatment guidelines). Table 1 summarizes the more detailed socio-demographic data of the participants, while Fig. 2 shows their distribution according to the various treatment regimens before GRT. Overall, the median (IQR) CD4 cell count at the time of failure was low, at 232.5 [161.25–401.25] cells/mm³. As to virological status, the overall median viral load (IQR) (global) was high at 54,480 [13,932.5–220,153] copies/mL. The detailed first- and second-line regimens are shown in Fig. 2.

Table 1
Socio-demographic and clinical factors.

	Frequency (n)	Percentage (%)
Age (years)		
0- 19	8	9.9
20- 29	10	12.3
30- 39	7	8.6
40- 49	35	43.2
50–59	12	14.8
≥60	9	11.2
Gender		
Female	47	58.0
Male	34	42.0
Residence		
Urban	63	77.8
Rural	18	22.2
Treatment line at failure		
First Line*	28	34.6
Second Line	53	65.4
Mutations		
NRTI	47	58
NNRTI	50	61.7
PI**	32	60.4
HIV subtype		
CRF02_AG	35	71.4
A1	5	10.2
F2	3	6.2
Others (CRF06_cpx, CRF13_cpx, G, G/A1)	6	12.2
Respected prescription		
Yes	75	92.6
Median viral load at failure, *copies/mL*	54,480 [13,932.5–220 153]	
Median duration on ART, *years*	10 [7–13]	
Median baseline CD4, *cells/mm³*	232.5 [161.25–401.25]	

* First line: We had 8 people failing a DTG-based and 20 failing NNRTI-based.
** n = 53 for PI-drug resistance mutations representing only those exposed to PI.

3.2. Acquired drug resistance patterns

The overall drug resistance rate (presence of at least one major DRM) was high at 50/81 (61.7%). The prevalence of drug resistance by antiretroviral class were as follows: nucleoside/nucleotide reverse transcriptase inhibitor (NRTI) resistance: 47/81 (58.0%); NNRTI resistance: 50/81 (61.7%; considering that all participants – even those found on DTG-based and PI-based at enrolment – had previous exposure to NNRTI as per our guidelines); and protease inhibitor (PI/r) resistance: 32/53 (60.4%) . Overall, the most prevalent mutations were M184V (24.6%), K103N (17%) and M46I (32.1%) respectively for NRTI, NNRTI and PI (see Fig. 3).

3.3. Prescribed regimens after GRT

From this study, we observed that the most commonly prescribed regimens among first line failures were AZT/3TC+LPVr &

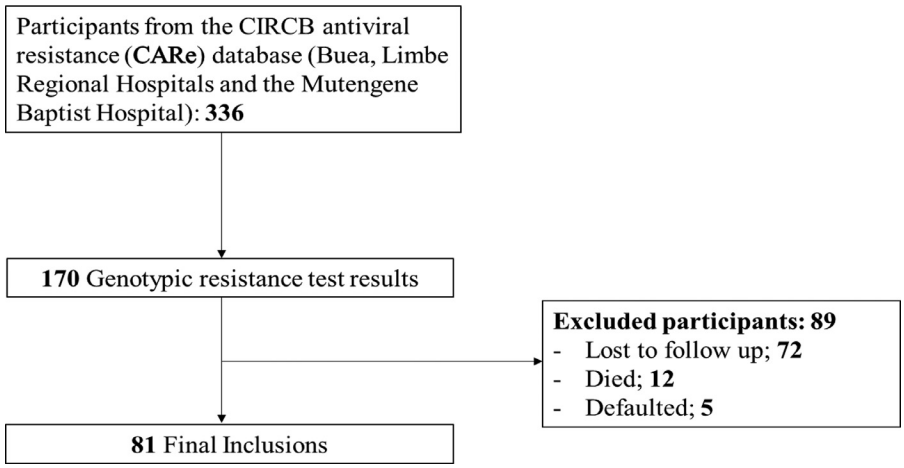

Fig. 1. Enrolment of participants into the study. NB: Medical records could not help us understand whether or not the deceased patients died because of disease progression or other causes. **Defaulter**: A patient actively stops medication or skips scheduled visits/sessions (e.g., skipping ART for a set period like 30+ days). **Lost to follow up**: A patient disappears from the healthcare system without a formal transfer, defined by missing a specified number of visits (e.g., two consecutive) or a duration (e.g., 2–6 months); these were patients for whom we could not trace treatment history or Post GRT follow-up data at any timepoint.

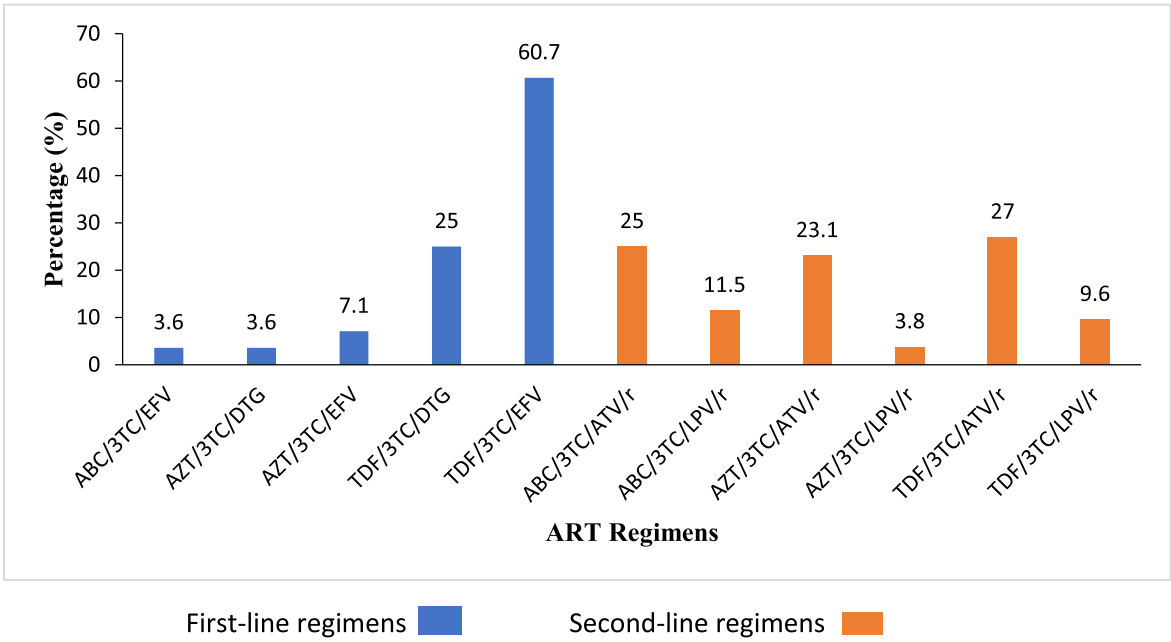

Fig. 2. Regimens prescribed before GRT. Efavirenz (EFV); abacavir (ABC); lamivudine (3TC); zidovudine (AZT); tenofovir (TDF); atazanavir boosted by ritonavir (ATV/r); lopinavir boosted by ritonavir (LPV/r); darunavir boosted by ritonavir (DRV/r); and dolutegravir (DTG).

AZT/3TC/ATV/r whereas we had TDF/3TC/DTG/DRVr among second line failures. Overall, the rate of DTG-containing prescriptions was 36.4% (including 24% being prescribed PI+INSTI) and DTG-sparing prescriptions was 63.6%. The details of the various prescribed regimens are shown in Fig. 4.

Data revealed that after first-line failures, about 39% of participants were maintained on first-line ART (28% on TDF+3TC+DTG and 11% on TDF+3TC+EFV), whereas about 43% were prescribed a second-line ART (LPV/r or ATV/r -based) and approximately 18% switched directly to a third-line ART (PI+INSTI based). Among those initially failing second-line ART, 7.6% were prescribed optimized first line ART (3.8% on AZT+3TC+DTG and 3.8% on TDF+3TC+DTG), 45.2% were maintained on a second-line ART (LPV/r or ATV/r -based) and approximately 47.2% switched to a third-line ART (PI+INSTI based).

3.4. Outcome after newly prescribed treatments

Overall, 92.6% (75/81) of the study participants respected the prescribed regimen. Following the prescribed treatments, available outcomes at 3, 6, 12, 24, or 36 months were collected. Specifically, 50.6% (41/81) of the participants had two post-GRT follow-up data; 35.8% (29/41) had three post-GRT follow-up data; and 13.6% (11/81) had four post-GRT follow-up data. Fig. 5 below presents virological response at these different timepoints.

Following WHO recommendations of viral load monitoring [24], participants' viral loads at 3, 6, 12, 24, and 36 months were recorded, highlighting respectively 35.0%, 56.5%, 64.4% 68.7% and 73.9% of undetectability; 45.0%, 28.3%, 17.0% 25.0% and 13.0% of low-level viremia (40–999 copies); translating a viral suppression of 80%, 84.8%, 81.4%, 93.7%, and 86.7%, respectively.

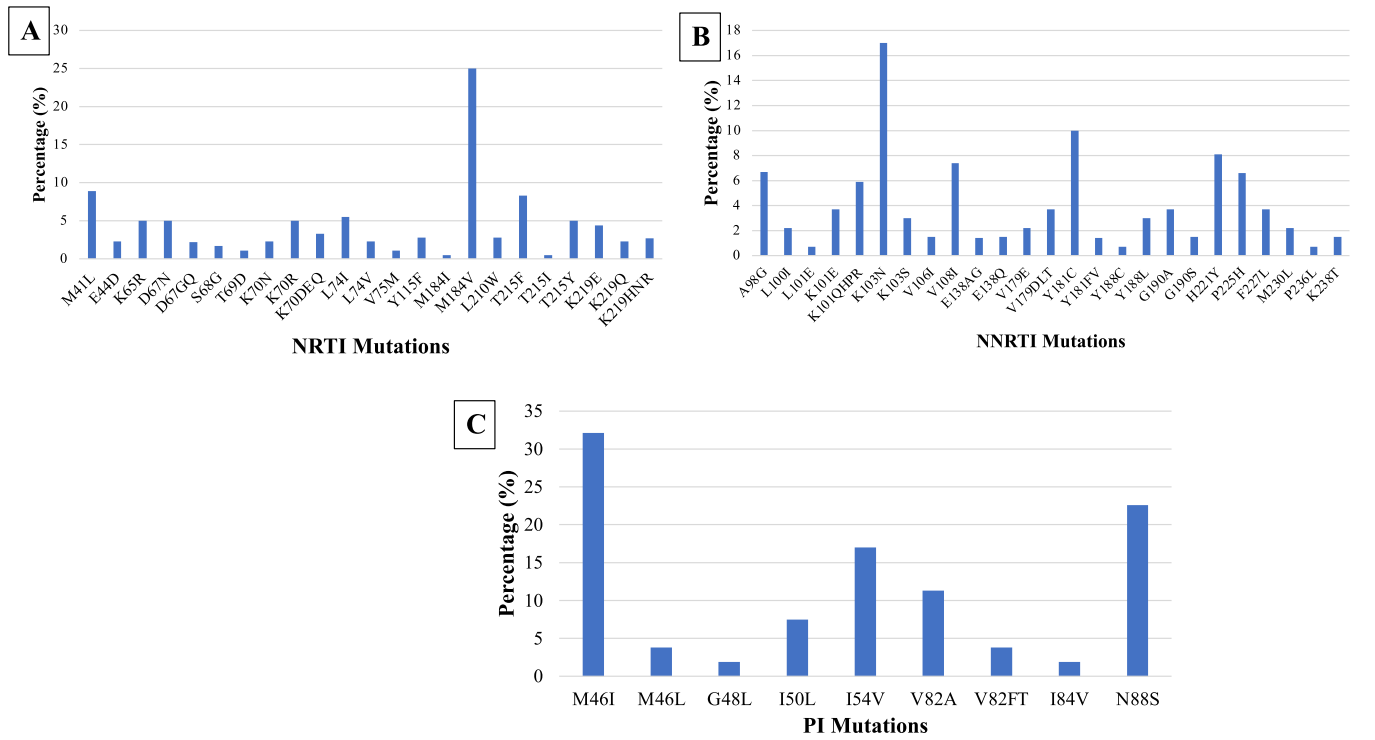

Fig. 3. Various resistance mutations by ARV drug class. Panel A: Resistance mutations to nucleotide reverse transcriptase inhibitors; Panel B: Resistance mutations to non-nucleotide reverse transcriptase inhibitors; Panel C: Resistance mutations to Protease inhibitors.

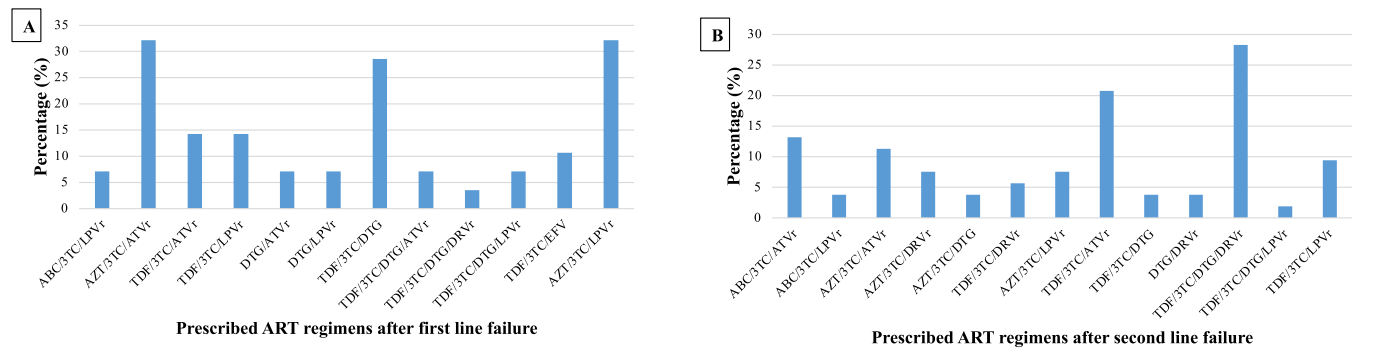

Fig. 4. Most prescribed regimens. Panel A: Prescribed regimens after first-line failure; Panel B: Prescribed regimens after second-line failure; efavirenz (EFV); abacavir (ABC); lamivudine (3TC); zidovudine (AZT); tenofovir (TDF); atazanavir boosted by ritonavir (ATV/r); lopinavir boosted by ritonavir (LPV/r); darunavir boosted by ritonavir (DRV/r); and dolutegravir (DTG).

3.5. Factors associated with the viral suppression

The association between viral suppression at the end of observation and potential associated factors was examined at a *P*-value cut-off of 0.05, with significance defined as $P \leq 0.05$. One-tailed Fisher's exact test was used for age distribution, gender, therapeutic line, duration on ART and genetic diversity, viral load and CD4 count.

Only the prescription of DTG-based regimens was found to be significantly associated to good virological response (i.e., viral suppression; VL < 1000 copies/mL). In effect, participants who received DTG-based were 3.21 times more likely to have suppressed viral loads.

3.6. Dynamic of the virological response according to time

In this study, we saw that DTG-sparing regimens were the most prescribed genotype-guided regimens, but they were associated with low viral suppression as seen in Table 2. Fig. 6 presents the dynamics of viral loads overtime according to regimens prescribed.

Interestingly, these data highlight the rapid ($P = 0.013$ at 3 months) and the long-term ($P < 0.05$ up to 24 months) effectiveness of DTG-based regimens compared to DTG-sparing regimens, translated by significantly greater virologic response in the DTG-based group. The observation was made respectively at 3, 6, 12, and 24 months. There was no statistical difference between virologic response on DTG-based compared to DTG-sparing regimens at 36 months post GRT.

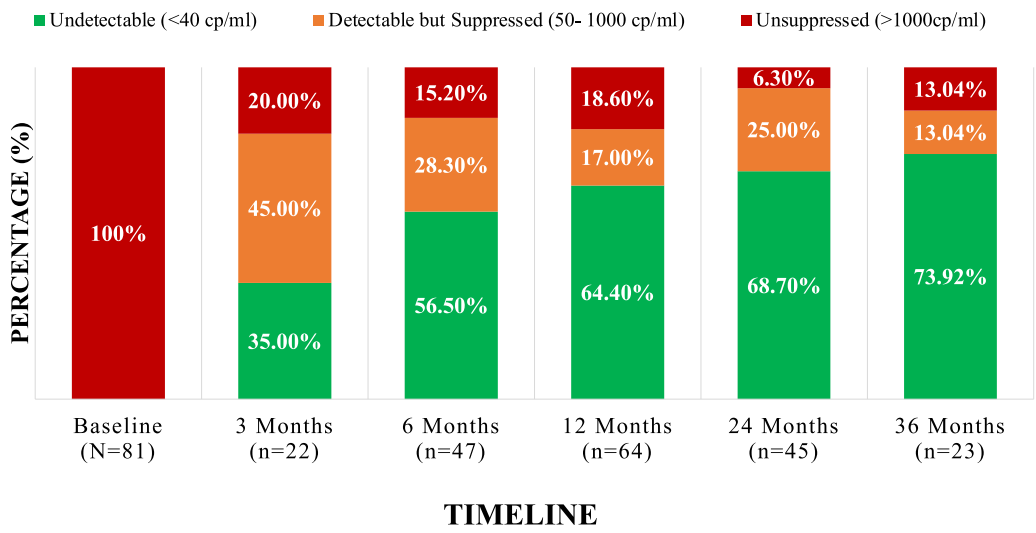

Fig. 5. Virological outcome following genotypic resistance test.

Table 2
Determinants of viral suppression in the study population.

Variables		Suppressed VL	Unsuppressed VL	*P*-value	Odds ratio [95%CI]
Age	≤43 years	31	11	0.058	0.32
	>43 years	35	4		[0.09–1.11]
Gender	F	41	6	0.10	2.46 [0.78–7.74]
	M	25	9		
Residence	Urban	54	9	0.07	3.00 [0.89–10.03]
	Rural	12	6		
CD4	≥232.5	21	3	0.64	0.95 [0.17–5.26]
	<232.5	22	3		
Viral load before GRT	≥54,480	33	7	0.47	1.21 [0.39–3.75]
(copies/mL)	<54,480	31	8		
Duration on ART	≥10 years	39	9	0.59	0.96[0.30–3.02]
	<10 years	27	6		
Treatment line before	First-line	24	4	0.34	1.57[0.45–5.48]
GRT	Second line	42	11		
DRM	Yes	42	8	0.32	1.53 [0.49–4.74]
	No	24	7		
Regimen respected	Yes	64	15	0.66	–
	No	2	0		
Genetic diversity	CRF02_AG	29	6	0.58	0.80 [0.14–4.57]
	OTHERS	12	2		
Type of regimen	DTG-based	45	6	**0.042**	**3.21 [1.01–10.21]**
prescribed	DTG-sparing	21	9		

4. Discussion

Antimicrobial resistance is a silent but serious health emergency threat worldwide. Antiretroviral strategy to fight against HIV in Cameroon, just as in many low-middle income countries (LMICs) follows a public health approach, thereby exposing highly treatment-experienced patients to multiple drug resistance mutations. However, access to genotypic resistance testing (GRT) is still limited in many LMICs, including Cameroon, where it is usually recommended only after failure of a second-line regimen [25]. This cohort study evaluated virological responses following GRT-guided treatment switches in PLWH residing in the South West region of Cameroon from 2018 to 2023. All age groups were included in our study, with a median age of 43 years. Thanks to the decreased mortality resulting from the effectiveness of ART, PLWH are now ageing, with their life expectancy increased by more than 50 years worldwide [26,27]. With respect to gender, there was a female predominance over males, reflecting data from the national birth registry in the country as well as previous studies [28,29]. Unfortunately, the above-mentioned increased life expectancy is most often jeopardized due to the decreasing effectiveness of ART subsequent to the emergence of acquired drug resistance mutations, which goes alongside longer treatment duration. Indeed, a study showed that individuals with a longer duration of ART exposure had overall higher rates of HIVDR and treatment failure as compared to those with a lower duration of treatment [30].

Our study population included people failing first- and second-line regimens before the GRT, with the majority failing protease inhibitor (PI/r)-based second-line therapies, with a median duration on treatment of 10 [7–13] years. In addition to this prolonged ART exposure, it is also well known that transitioning from first- to second-line regimens without the help of GRT in LMICs would lead to failure on second-line therapies, possibly due to the accumulation of NRTI mutations and potential cross-resistance among NRTIs [31]. Our observations are thus consistent with evidence from long-term follow-up cohort in Vietnam demonstrating a marked increase in the number and complexity of drug-resistance mutations, with NRTI resistance approaching 100% and NNRTI efficacy declining to nearly zero [32]. These results altogether clearly illustrate how prolonged exposure to suboptimal/failing regimens accelerates the accumulation of HIVDR, thereby reducing future therapeutic options especially in LMICs.

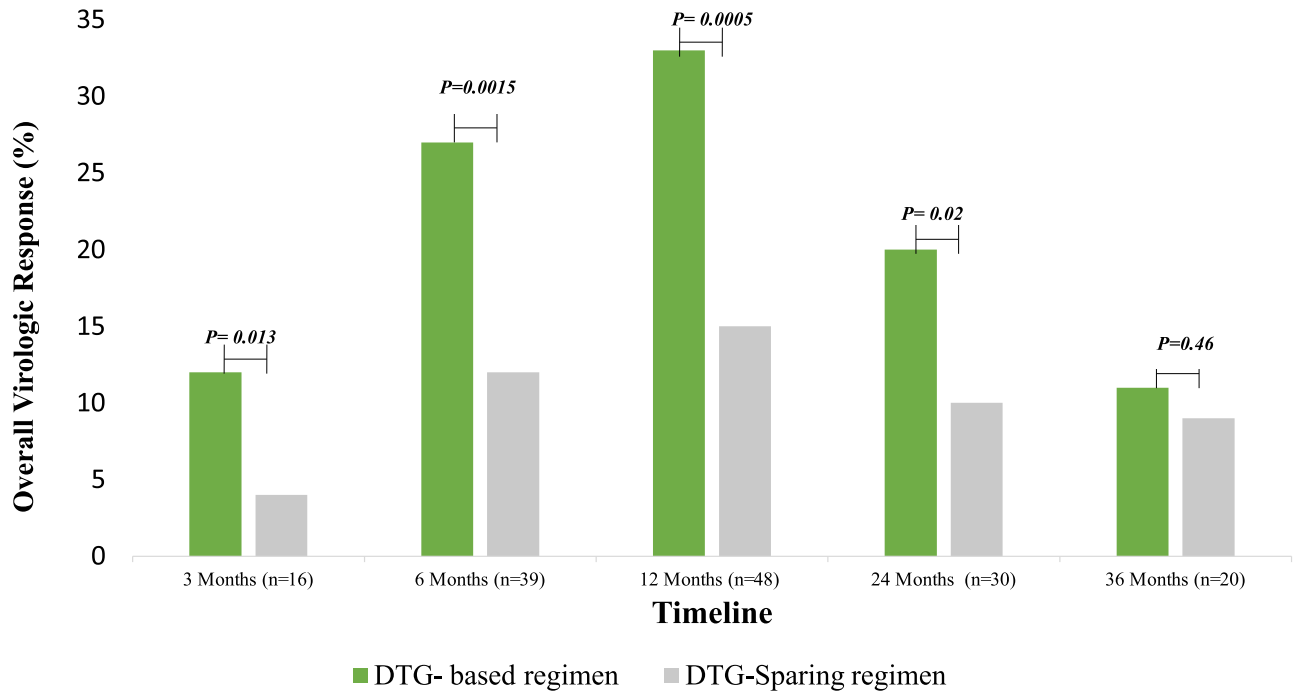

Fig. 6. Significance of DTG-based versus DTG-sparing regimens overtime.

This strengthens the clinical value of timely prescription of GRT-guided switch, as applied in our study, to prevent the rapid emergence of HIVDR and preserve salvage therapeutic options on the long run. Furthermore, the immunovirological status at ART failure in the current study was poor, with a high median viral load and a low median CD4 count. Also, the rate of HIVDR was 61.7%, with NRTI, NNRTI, and PI/r resistance being 58.0%, 61.7% and 60.4% respectively, suggesting not all participants enrolled actually harbored HIVDR; some only needed reinforced adherence with their respective regimens [33,28,34]. Notably, TDF/3TC/DTG, TDF/3TC/DTG/ATV/r and ATV/r + 2NRTI combinations were frequently prescribed for both first- and second-line failures whereas DTG+DRV/r + 2NRTI was predominantly prescribed after second line failures. It is also worth noting that many of these common regimens were reused translating preserved regimen efficacy and justifying adherence reinforcement (specifically among those without resistance) while very few individuals (found without drug resistant mutations) were continued on TDF/3TC/EFV combination; which was still active. Furthermore, our dataset clearly establishes sustained viral suppression (VL<1000 copies/mL) and even viral undetectability (VL<50 copies/mL) over 3 years following GRT-guided switch in individuals with multiclass HIVDR. Even though this is unsurprising for high-income settings where patients are being initiated following recommendations from GRT [14,32,35], the context in LMICs will limit the broad implementation of such measures, considering the cost that will be incurred. Nonetheless, this important result could not have been possible if compliance to treatment was not achieved. In effect, the data revealed about 93% of the participants respecting GRT-recommended prescriptions; which in turn translates an improved adherence. Specifically, a good virological response was obtained as soon as 3 months post-GRT, which is a relatively short timeframe. Impressively, this rapid response was sustained with growing levels of viral undetectability (VL<50 copies/mL) up to the end of the observation (from ~35% at 3 months to >73% after 36 months); translating an encouraging trend for disease control when re-enforced adherence is combined with optimized regimens [36]. Similar observations were previously made in France and Cameroon, emphasizing the usefulness of GRT for treatment optimization [11,31,34,37]. Importantly, though the overall virological response was improved with genotype-guided regimens, it is also worth noting that these data highlight the rapid ($P = 0.013$ at 3 months) and the long-term ($P < 0.05$ up to 24 months) effectiveness of DTG-based regimens compared to DTG-sparing regimens. On further notice, DTG-based regimens (DTG+PI/r in most cases) globally led to sustained viral suppression/viral undetectability compared to DTG-sparing regimens up to 24 months after which we observed a decrease in the rate of viral suppression (from 94.7% at 24 months to 86.9% at 36 months). Although the 'DTG+DRV/r' combination has been proven to yield very good virological responses in such clinical scenarios previously [11,36], the virological rebound observed among suppressed but detectable PLWH between 24 months and 36 months post-GRT calls for in-depth interpretation and investigation. Nevertheless, this result, alongside global policy reviews by Nutt *et al.* (2025) and long-term resistance surveillance data by Hung *et al.* (2025), emphasizes the benefits of optimized core drugs with very high potency and a genetic barrier, such as DTG [32,35]. Our findings are therefore very encouraging for ART-guidelines in LMICs and beyond as clinical practices usually report viral load reduction followed by virological rebounds after treatment-change. This is all the more relevant as we later observed rapid and long-term efficacy obtained despite advanced immune-virological data before GRT.

As study limitations, some DTG-failures (08 cases) were classified as first-line failures despite prior exposure to efavirenz-based; this was done in order to align to our national guidelines, which classify them as transitions to an optimized first-line ART and not as switches to second-line ART. However, such cases may have had little or no impact on the overall outcome of second-line failures post-GRT. Additionally, the reasons for virological rebound observed among some individuals between the second and third years post-GRT have not been clearly elucidated. However, this will be addressed in a subsequent study encompassing a larger sample size. Finally, we did not have INSTI resistance data in our study

population because majority of the study population lacked integrase genotyping due to the fact that they were enrolled between 2018 and 2021 when the assay was not yet a routine locally.

5. Conclusions

In a nutshell, this study reports a sustained viral suppression over 3 years among individuals with multiclass HIV drug resistance following genotypic-guided treatment change in Cameroon. Importantly, dolutegravir-based regimen prescriptions yielded a higher rate of viral suppression (VL < 1000 copies/mL) and virological control (VL < 50 copies/mL), which was consistently observed after 2 years. Overall, these findings underscore the necessity of integrating affordable and scalable models of ART strategies that combine reinforced adherence with treatment optimization (through genotypic resistance testing) into LMIC programs, to sustain global progress towards UNAIDS's 'third 95′ target and achieve an epidemic control by 2030, as ambitioned.

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