

Predicting Virological Failure Among HIV Patients Using Machine Learning in Cameroon, 2023-2024

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ABSTRACT

Background: Sub-Saharan Africa continues to shoulder the heaviest burden of the global HIV epidemic and Cameroon is not an exception, nearly half a million people are living with the virus, and treatment programmes still struggle to keep virological failure below the 10% ceiling recommended by WHO. Part of the problem is viral load testing that is done only periodically, so when treatment starts to fail, ART clinics often don't find out until three (3) to six (6) months after real damage has already been done, resistance has built up, and the patient has been quietly slipping through the cracks for months. Traditional statistical approaches have provided insights, but they often struggle with complex, non-linear predictors. Machine learning offers new opportunities to identify patients at risk of failure using data already collected in routine care as has been done in Ghana, Nigeria, and elsewhere. This study explores how similar approaches can be applied using data from Cameroonian ART clinics.

Methods: Data from 1,968 anonymized ART patients enrolled between January 2023 and December 2024 at Nkwen Baptist Hospital and Bafoussam Baptist Hospital were retrospectively collected, pre-processed with imputation of missing values, exclusion of variables with extreme missingness, and balancing of outcome classes using SMOTE. Four supervised algorithms: Logistic Regression (LR), Random Forest (RF), Support Vector Machine (SVM), and Gradient Boosting Machine (GBM) were trained with cross-validation. Model interpretability was enhanced using SHAP values to identify the most influential predictors.

Results: All models achieved comparable accuracy (~70%), but the gradient boosting model performed best, correctly identifying 80% of patients who went on to fail treatment. SHAP analysis revealed that body weight and age were the strongest predictors, surpassing sex and clinic site in importance. This interpretability is critical, as it allows clinicians to understand why the model flags certain patients and to integrate predictions into routine decision-making.

Conclusion: This study demonstrates that machine learning can provide clinically interpretable predictions of virological failure using routinely collected data in Cameroon. By flagging high-risk groups, such models could complement viral load monitoring, enable earlier interventions and reduce the risk of drug resistance. With further validation, predictive tools of this kind could strengthen ART programs and support progress toward the UNAIDS 95-95-95 targets. These models can help shift HIV care from reactive detection of failure toward proactive prevention.

Keywords: Virological Failure; HIV; Antiretroviral Therapy; Machine Learning; Shap; Cameroon; Predictive Modelling

Introduction

The Human Immunodeficiency Virus (HIV) is a lentivirus that targets and progressively destroys CD4 T lymphocytes, the cells that coordinate the body's immune response. When left untreated, this decline eventually leads to acquired immunodeficiency syndrome (AIDS), the advanced stage of infection marked by severe immunosuppression and opportunistic disease [1,2]. Phylogenetic evidence indicates that HIV-1 emerged zoonotically from simian immunodeficiency virus (SIVcpz) circulating in chimpanzees, most likely through a cross-species transmission event in southeastern Cameroon in the early twentieth century, while HIV-2 arose independently from SIV infecting sooty mangabeys in West Africa [3,4]. The resulting disease was first recognized clinically in 1981 amid unusual clusters of opportunistic infections in the United States, and the causative virus was isolated two years later by Françoise Barré-Sinoussi and Luc Montagnier at the Institut Pasteur work later honored with the 2008 Nobel Prize in Physiology or Medicine [5,6]. HIV spreads through direct contact with blood, semen, vaginal or rectal fluids, or breast milk from an infected person, chiefly via unprotected sexual contact, the sharing of needles or transfusion of unscreened blood products, and mother-to-child transmission during pregnancy, delivery, or breastfeeding [7,8]. It is not transmitted through casual contact, and a person on effective antiretroviral therapy (ART) with a sustained undetectable viral load cannot sexually transmit the virus, a principle summarized as "Undetectable = Untransmittable" [9].

Without treatment, progressive immune decline leaves patients vulnerable to AIDS-defining opportunistic infections and malignancies such as tuberculosis, *Pneumocystis pneumonia*, and Kaposi's sarcoma [8,5]. Beyond individual morbidity, HIV remains a leading cause of adult mortality in heavily affected regions, driving household impoverishment and child orphanhood, and continues to be compounded by stigma that delays testing and care seeking [8]. More than 44 million lives have been lost to AIDS-related illness since the epidemic began, underscoring the continued urgency of early diagnosis, sustained ART, and predictive tools capable of pre-empting treatment failure which is the focus of this study [8]. Human Immunodeficiency Virus (HIV) infection remains one of the defining public health challenges of the twenty-first century. Globally, an estimated 40.8 million people are living with HIV, 31.6 million of whom are receiving antiretroviral therapy (ART); 1.3 million new infections and 630,000 AIDS-related deaths were recorded in 2024 alone [8]. Sub-Saharan Africa continues to bear the greatest share of this burden, accounting for roughly 65% of all people living with HIV worldwide, with 5.2 million cases and nearly 4 million individuals on ART in Western and Central Africa alone. In response, the Joint United Nations Programme on HIV/AIDS (UNAIDS) has progressively raised its global targets from 90-90-90 to the more ambitious 95-95-95 framework for 2030, underscoring that viral suppression not simply treatment coverage is the ultimate benchmark of programmatic success [8-10].

Cameroon exemplifies both the scale and the complexity of this challenge. National HIV prevalence stands at 2.6%, with close to half a million people living with the virus and approximately 438,000 adults currently on ART; roughly 7,600 HIV-related deaths were recorded in 2024. Within this context, virological failure (VF) commonly defined as two consecutive viral load measurements exceeding 1,000 copies/mL after adequate treatment duration despite adherence support has emerged as a critical and persistent threat to treatment success [11,9,12]. The World Health Organization stipulates that VF should affect no more than 10% of patients on ART [13]; yet empirical evidence tells a very different story. Pooled estimates across sub-Saharan Africa place VF prevalence between 18% and 30% among adults on first-line therapy [12-14] while Cameroon-specific studies report rates ranging from 10% to 30% [15-17]. The clinical and public health consequences of VF are substantial. At the individual level, unresolved viral replication accelerates CD4 decline, increases susceptibility to AIDS and non-AIDS-related comorbidities, and raises mortality risk two to three fold relative to virally suppressed patients [18]. Drug resistance mutations (DRMs) emerge in an estimated 40-60% of VF cases, frequently necessitating a costly and less tolerable switch to second- or third-line regimens [19-21]. At the population level, unchecked VF undermines the third "95" of the UNAIDS targets, sustains onward transmission, and disproportionately affects children and other vulnerable groups [22,23].

The determinants of VF are multifactorial, spanning sociodemographic factors (age, sex, marital status), clinical characteristics (low BMI, tuberculosis co-infection), treatment related factors (regimen type, dosing frequency, drug stockouts), and health systems constraints such as delayed viral load turnaround time and inconsistent monitoring, because these predictors interact in complex, often non-linear ways and vary considerably across settings, no single risk factor reliably identifies patients at risk before failure occurs [24]. This complexity has motivated a shift toward computational approaches capable of modeling high-dimensional, interacting clinical data. Traditional statistical techniques, such as logistic regression, remain valuable for their interpretability but are constrained by assumptions of linearity and limited capacity to accommodate missing data or complex variable interaction [25-27]. Machine learning (ML), by contrast, can uncover non-linear patterns across large and heterogeneous datasets without requiring such assumptions to be specified in advance [28-30]. Algorithms such as random forests, gradient boosting machines, and support vector machines have already demonstrated strong predictive performance across a range of healthcare applications, including disease diagnosis, mortality forecasting, and treatment outcome prediction [31,32]. Within HIV care specifically, ML models have been used to predict adherence, loss to follow-up, and virological outcomes, at times outperforming conventional statistical methods; for example, a random forest model developed in Ethiopia achieved high predictive accuracy for virological failure [33], while comparable applications have since been reported in Ghana and Nigeria, to name a few [34-37].

A recurring concern with ML, however, is its “black box” nature: the internal logic driving individual predictions is often opaque, which can erode clinician trust and slow adoption in settings where transparency is essential to safe decision making [38-41]. Explainable artificial intelligence (XAI) techniques—most notably Shapley Additive Explanations (SHAP) and feature importance ranking—have been developed to address this limitation by clarifying how individual predictors shape model output, thereby bridging the gap between predictive accuracy and clinical usability [41,42]. Despite this progress, the application of ML-explainable or otherwise-to HIV outcomes in Cameroon remains almost entirely unexplored. As of January 2026, no peer reviewed study has reported the development or validation of a machine learning model to predict virological failure in the Cameroonian context, even as comparable efforts elsewhere in West and Central Africa demonstrate clear feasibility and clinical value [33,35]. This gap is not merely academic. In Cameroon, Virological Failure (VF) among HIV patients is currently detected reactively, only after elevated viral load has been confirmed through periodic testing. Viral load monitoring, conducted every 6 months for stable patients or every 3 months in special circumstances, is the sole determinant of Antiretroviral Therapy (ART) follow up and suppression. This reliance on scheduled viral load assessments means that VF may remain undetected for extended periods between tests, during which patients can accumulate drug resistance mutations and experience clinical deterioration.

The absence of proactive or predictive monitoring tools delays timely intervention, thereby limiting opportunities to optimize treatment, reduce resistance, and improve patient outcomes. Routine monitoring is constrained by infrastructural limitations, incomplete patient records, and delays in viral load turnaround, all of which allow patients to remain on failing regimens longer than clinically desirable [43,44]. If the risk of VF could instead be estimated at or shortly after treatment initiation—using data that health facilities already routinely collect—clinicians would be able to intervene earlier: intensifying adherence counselling, reassessing regimens, or prioritizing high risk patients for closer follow up, before resistance accumulates or transmission occurs. Machine learning is well suited to this task precisely because it can integrate sociodemographic, clinical, laboratory, and adherence related data simultaneously, capturing interactions among predictors that traditional regression-based models are ill equipped to model [45-47]. Yet the performance of any predictive model is inherently context-dependent: predictors, base rates, and data quality in Cameroonian ART clinics differ from those in the Ethiopian, Ghanaian and other settings where most prior ML VF research has been conducted [19,33,34,37]. A model developed and validated elsewhere therefore cannot be assumed to generalize reliably to Cameroon. It is this locally grounded, unaddressed need for a context specific, interpretable, and validated predictive tool that motivates the present study. By leveraging routinely collected register data from two ART clinics in Cameroon’s North West and West Regions, this research seeks not only to fill an evidence gap but to generate a practical deci-

sion support tool that can inform differentiated care and strengthen progress toward the UNAIDS 95-95-95 targets. This study was therefore guided by the following research question:

How accurately and interpretably can machine learning models, trained on routinely collected clinical and demographic data from ART clinics in Cameroon, predict virological failure among adult HIV patients?

Methodology of Study

Study Area

This study employed a retrospective cross-sectional study which was carried out at two major ART centers in Cameroon notably Nkwen Baptist Hospital in Bamenda (North West Region) and Bafoussam Baptist Hospital in the West Region. Both facilities operate under the Cameroon Baptist Convention Health Services (CBCHS) network and serve diverse urban and peri-urban populations. They are also supported by PEPFAR funded HIV programs, making them key hubs for HIV care and treatment in the country.

Study Population

The study included all adult HIV positive patients (≥ 18 years) who initiated ART at either hospital. To be eligible, patients needed to have at least one follow up viral load (VL) measurement taken six months or more after starting therapy in either hospital.

Study Duration

Data were retrospectively extracted for ART initiations and follow up visits covering the period from January 2023 through December 2024.

Sampling Method

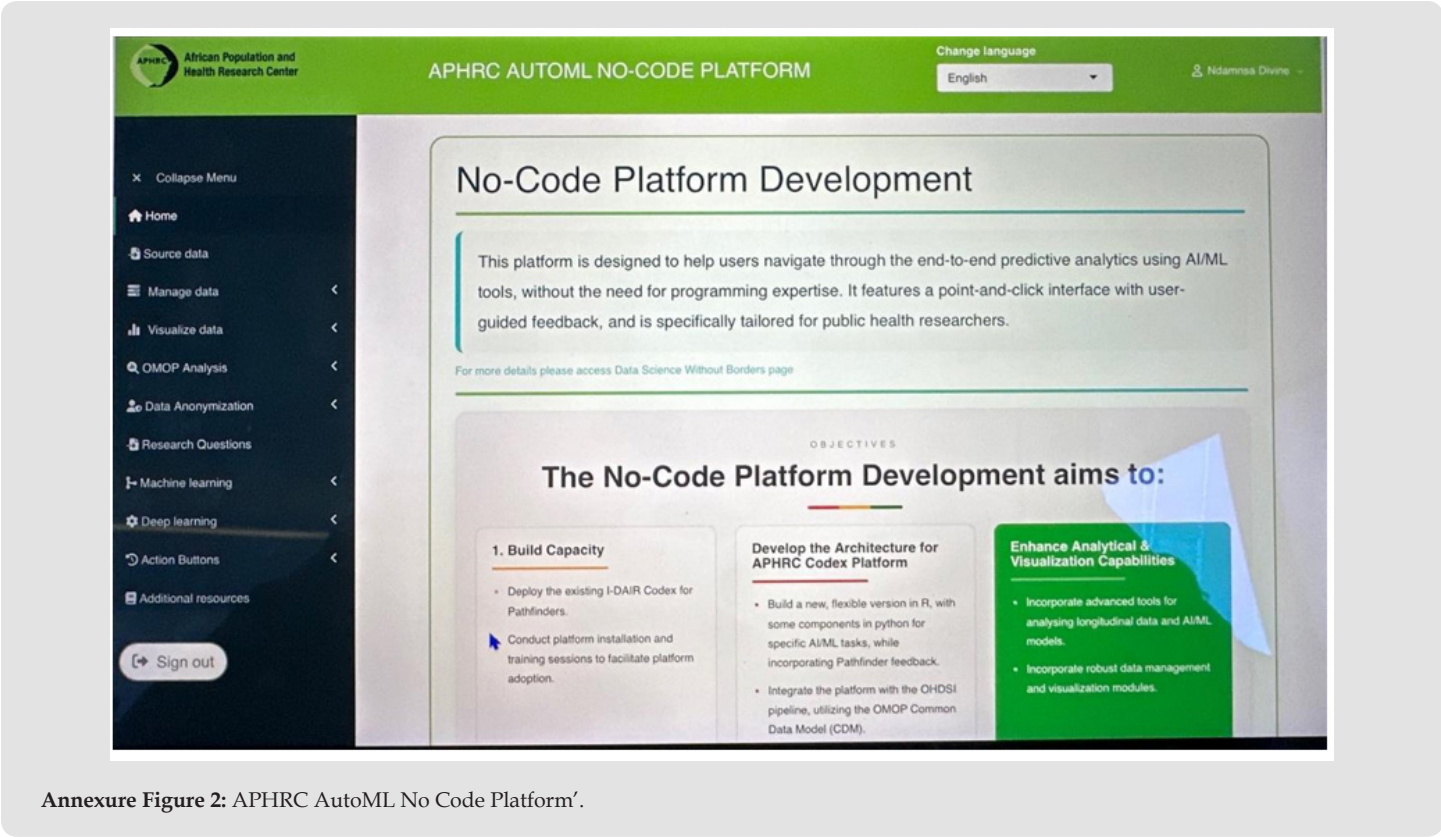
A census approach was adopted, meaning all patients who met the inclusion criteria during the study period were considered. Patients were excluded if they had missing baseline data, were lost to follow up before six months, or lacked at least one VL result.

Study Tools

Data extraction followed a standardized checklist (Annex 1), which captured demographic, clinical, laboratory, and adherence related variables from patient registers. Patient level data obtained from the ART registers, with all personal identifiers were removed to ensure confidentiality as per ethics. After extraction, data cleaning, feature engineering, and machine learning model development were conducted using APhRC AutoML No Code platform (Annex 2). All analyses adhered to standardized variable definitions, including the operational definition of VF as a viral load $>1,000$ copies/mL after at least six months on ART. Hyperparameter tuning was carried out using algorithm specific grids informed by established best practices. Model selection relied primarily on the AUC ROC metric, chosen for its robustness in handling class imbalance and independence from threshold effects.

Annexure 1: Data Extraction Checklist.

Re-gion	Dis-tract	Site Name	study id	Sex	Age	art start date	ARV Regi-men	Result date	v1 results	CD4	dispen-sation site	Adher-ence classification	adher-ence proxy	Profes-sion	weight kg	Height cm	bmi	Clin-ical Stage



Annexure Figure 2: APHRC AutoML No Code Platform’.

Study Size

The sample size for this study was determined using the Rule of Thumb for ML (10 Event Per Variable, EPV = 10), which is recommended for predictive modeling and machine learning studies to ensure adequate outcome events relative to the number of predictors and to minimize model overfitting. Methodological guidelines suggest 10–20 outcome events per predictor variable. This study anticipates including approximately 10 predictor variables (e.g., age, sex, CD4 count, viral load, ART regimen, adherence status, WHO clinical stage, duration on ART, etc). Using a conservative estimate of 12 events per

variable (VF prevalence of ~12%), a minimum of 120 virological failure events was required. Given an estimated 12% prevalence of virological failure among patients on ART in Cameroon, the minimum total sample size required to obtain 120 events was calculated as:

Total Sample=120/0.12=1,000

To enhance model robustness and account for incomplete records, a larger dataset was used. The study employed a retrospective analysis of 1,968 dataset containing epidemiological information on HIV treatment follow up, demographic, adherence, laboratory and clinical data and a total of 18 variables shown in Table 1 below.

Table 1: Variable Distribution.

Variable Category	Specific Variables	Source
Demographic	Region – character	Patient registration records
	District – character	
	Site name – character	
	Serial number – character	
	Age – continuous	
	Sex – character	
	occupation – character	
	dispensation site – character	
Clinical	Weight – numeric	Clinical assessment forms
	Height – numeric	
	WHO clinical stage (excluded) – character	
	BMI (excluded) – numeric	
Laboratory	Baseline and/or follow up CD4 count – continuous	Laboratory records, viral load database
	viral load results – binary	
Treatment	ART regimen – character	Pharmacy and treatment registers
	duration on ART – numeric	
Adherence	Adherence Proxy – numeric	Pharmacy refill and consultation records
	Adherence Classification – character	
	(pharmacy refill records, missed visits, missed consultations)	

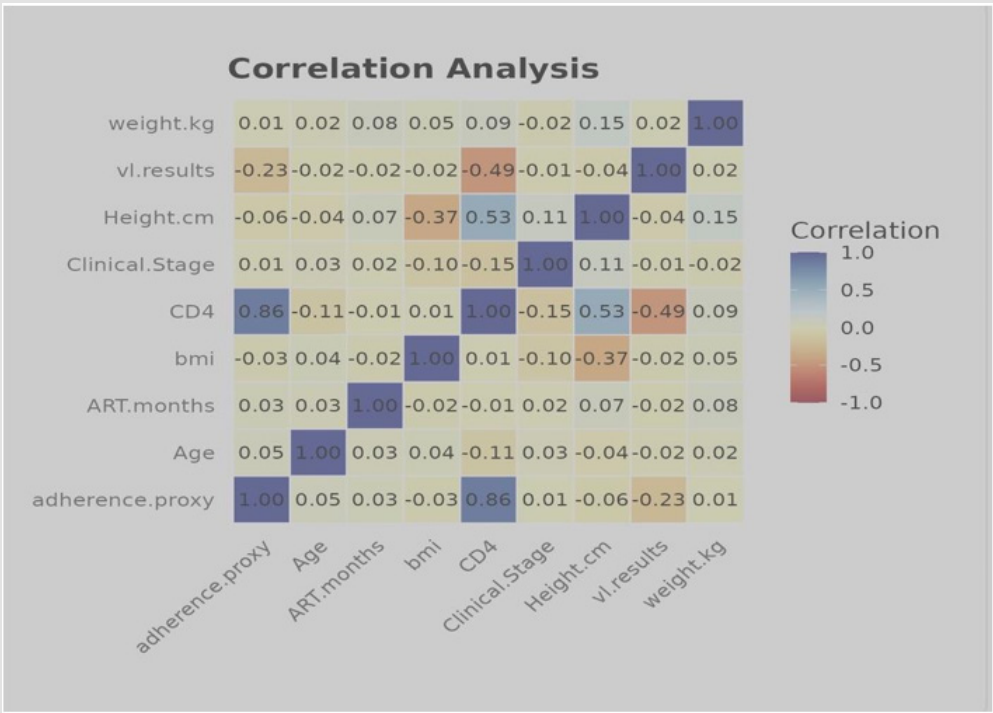
Outcome Definition

Virological Failure was the primary outcome of interest, defined according to WHO criteria as a plasma HIV viral load $\geq 1,000$ copies/mL at the most recent measurement. For analysis, this outcome was treated as a binary variable (Failure vs. Suppressed). Adherence proxy was derived from routinely collected patient data, which included pharmacy refill records (calculated on 100%), documented missed drug doses (calculated on 100%), and missed clinical consultations (calculated on 100%). Patients were classified as having suboptimal adherence if any of these indicators suggested non adherence during follow up (calculated as total of all three values divided by 3), while those with timely pharmacy refills, no reported missed doses, and no missed consultations were classified as adherent. Adherence classification was then employed in terms of classes as shown below

Data Preprocessing

A number of preprocessing steps were undertaken to prepare the data set for modelling. To ensure a robust model performance, some variables were excluded for their irrelevance such as region, district, serial number; missing values were equally identified and handled within the recipes preprocessing pipeline: numeric variables

were imputed using the median, and categorical variables using the most frequent category, with both steps estimated exclusively on the training data and then applied unchanged to the test data to prevent information leakage. Variables with disproportionately high missingness-CD4, height, BMI, WHO clinical stage -were excluded from the final models rather than imputed, on the basis that extensive imputation of these variables was judged more likely to introduce bias and noise than to add predictive value given the other clinical variables retained, whereas weight_kg and art. months were retained due to a low missingness percentage of about 31.25% and 4.776% respectively and they were median imputed. The final set of predictor variables included: age, sex, site_name, weight_kg, art_month, adherence_classification, adherence_proxy, arv_regimen, dispensation_site “viral_status” serving as the binary outcome variable (1 = failed, 0 = suppressed). Further preprocessing analysis unveiled CD4, adherence classification and adherence proxy exhibiting strong multicollinearity with other predictors in the dataset. Specifically, CD4 and adherence proxy were highly correlated (annexe 3), meaning they carry overlapping information that risks inflating variance and destabilizing model estimates. To reduce redundancy, avoid biased coefficients, and improve the robustness of the predictive model, these variables were removed (Table 2).



Annexure Figure 3: Correlation Analysis.

Table 2: Missingness Percentage.

variable missing	Percentage
CD4	96.44%
Profession	93.14%
bmi	68.24%
Height_cm	67.73%
Clinical_Stage	43.39%
Weight_kg	31.25%
Art_months	4.78%
Adherence_classification	0.00%
serial	0.00%
Site_Name	0.00%
Study_id	0.00%
Sex	0.00%
Age	0.00%
ARV_Regimen	0.00%
vl_status	0.00%
Dispensation_site	0.00%
Adherence_proxy	0.00%

Ethical Considerations

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki and the national research ethics guidelines of Cameroon. Ethical clearance was obtained from the Cameroon Baptist Convention Health Services Institutional Review Board (CBCHS IRB) prior to the commencement of data collection, under approval reference IRB2025 110, covering the study protocol titled “Predictive Analysis of Virological Failure among HIV Patients on Antiretroviral Therapy Using Machine Learning Approaches”, valid for the period 12 November 2025 to 12 November 2026. Site level authorization to conduct data collection was subsequently confirmed in writing by hospital administration at each participating facility; for Bafoussam Baptist Hospital, this authorization is documented under reference CBCHS/BaBH/Adm/L 25/472 (10 December 2025). Because the study relied exclusively on routinely collected, retrospective patient register data rather than direct patient contact or intervention, the CBCHS IRB granted a waiver of individual informed consent. All data were extracted after removal of personal identifiers, and patients were assigned anonymized study codes prior to analysis, so that no individual could be identified from the dataset used for model development. Data were stored on password-protected devices accessible only to the research team, and were used strictly for the purposes described in the approved protocol. No aspect of routine clinical care was altered as a result of this research, and findings are reported at the aggregate, de-identified level throughout this study.

The dataset was randomly partitioned into training (80%) and test (20%) subsets using stratified sampling to preserve the original outcome distribution in both subsets; the test set was reserved exclusively for final model evaluation.

Four supervised ML algorithms were developed and trained on this data set:

1. logistic regression (glmnet) a regularization framework that performs variable selection and shrinkage [48];
2. Random Forest an ensemble tree-based algorithm that combines multiple decision trees to improve predictive accuracy [49];
3. Support Vector Machine with a radial kernel a classification algorithm that identifies optimal decision boundaries in high-dimensional feature space [50]; and
4. Gradient Boosting an efficient gradient boosting algorithm widely used for high performance predictive modeling [51].

All models were trained within the caret framework in APHRC AutoML No Code Platform [52-54], using repeated 10 fold cross-validation (three repeats) with grid search hyperparameter tuning optimized against the area under the receiver operating characteristic curve (AUC ROC), chosen for its robustness to class imbalance and independence from a fixed classification threshold. Class imbalance in the training data was addressed using SMOTE, applied within each cross-validation fold [55]. Because the primary aim of the study was prediction rather than causal effect estimation, confounding was not addressed through traditional stratification or covariate adjusted regression; instead, all candidate predictors were entered simultaneously into each multivariable ML model, allowing the algorithms to account for correlated and potentially confounded predictor relationships directly, with the SHAPLEY values subsequently used to identify robust, non-redundant predictors and to guard against attributing importance to spurious or confounded associations. All analyses were conducted in the No Code Platform, using the caret, with session metadata archived to support reproducibility [56-59] (Figure 1).

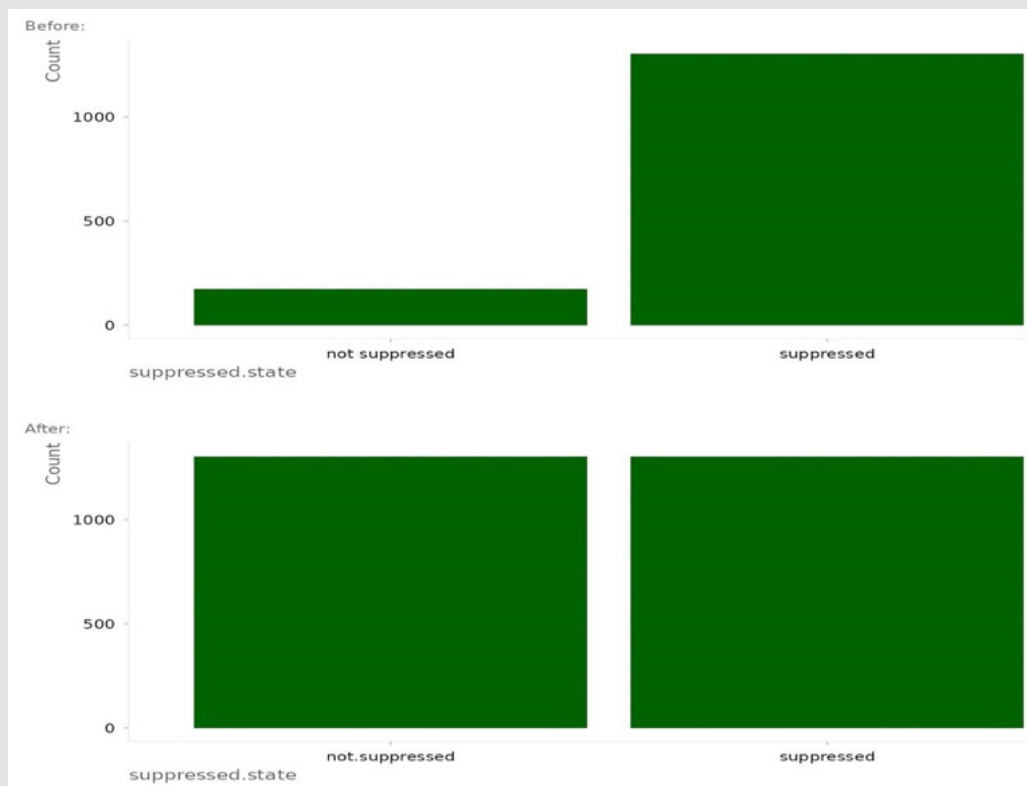


Figure 1: Class Balance Before and After Model Balance with SMOTE.

Subgroups and Interactions

Some subgroup or interaction analyses were analysed as secondary, some differences in predicted risk observed across demographic strata during the interpretation of results (for example, an apparent concentration of risk among younger male patients) emerged as exploratory and should accordingly be interpreted as hypothesis generating rather than confirmatory. Several design features were intended to limit common sources of bias. To limit bias, the study included all

eligible patients to avoid selection bias, used a standardized checklist to reduce measurement errors, applied predefined imputation and excluded variables with extreme missingness to handle data gaps, held out 20% of data and isolated preprocessing to prevent overfitting, balanced the minority VF class with SMOTE, and applied SHAP explanations to improve interpretability and clinician trust. In order to run the model, there is a predefined pathway to follow defined below (Figure 2).

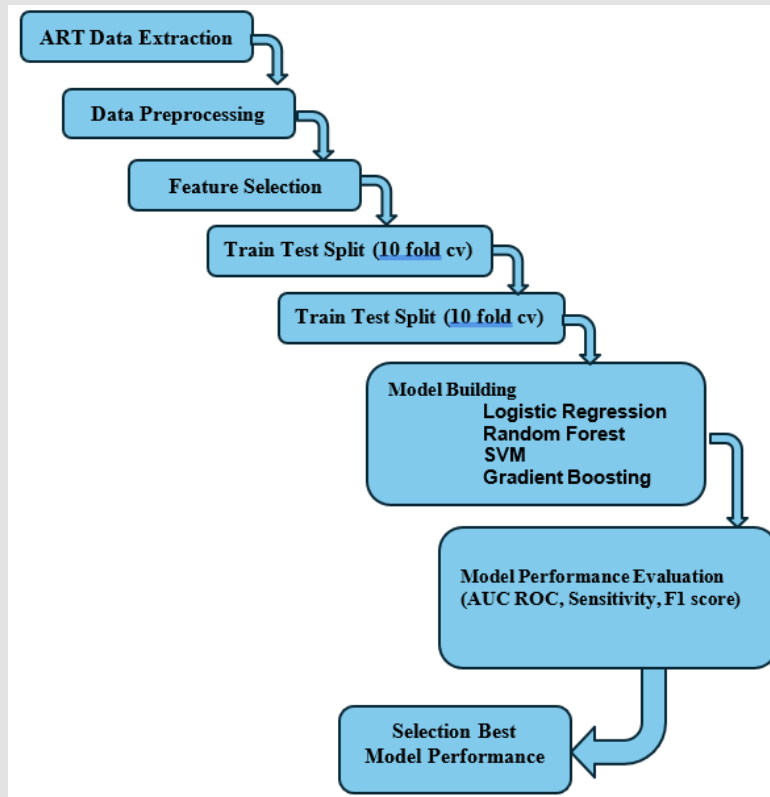


Figure 2: Study Flow Diagram.

Model Validation

Cross Validation and Feature Selection

Model development incorporated 10 fold cross-validation to strengthen generalizability and reduce the risk of overfitting. Feature selection was guided by the Boruta algorithm, with SHAP values used to complement this process by enhancing interpretability and highlighting the most influential predictors. Comparative evaluation across candidate models ensured a systematic approach to selecting the best performing algorithm for final analysis.

Model Tuning and Selection

Hyperparameters were optimized using grid search within the

cross-validation framework. The area under the receiver operating characteristic curve (AUC) served as the primary optimization metric, chosen for its robustness in handling class imbalance and threshold independence. Final models for each algorithm were selected based on their cross-validated performance.

Model Evaluation

Performance was assessed on the held-out test dataset using a comprehensive set of metrics: AUC, accuracy, sensitivity, specificity, precision, and F1 score. Binary classification thresholds were fixed at 0.5 to maintain consistency across models. Confusion matrices were generated to derive performance measures, while Receiver Operating Characteristic (ROC) curves provided comparative visualization of predictive capability.

Model Interpretability

To improve transparency, Shapley Additive Explanations (SHAP) were applied. Global feature importance and directional effects were visualized through SHAP summary and dependence plots, offering clear insights into the predictors most strongly associated with virological failure risk.

Results

Baseline Characteristics and Virological Failure

A total of 1,968 adult patients-initiated ART between January 2023 and December 2024 met the study’s inclusion criteria. The median age was 43 years (range: 18-92), and the participants were predominantly female (71.8%). Most participants were enrolled at Nkwen Baptist Hospital (72.4%), while the remainder were from Bafoussam Baptist Hospital (Table 3). The overall prevalence of Virological Failure (VF) was 11.7%, a figure consistent with estimates reported from comparable ART programs across sub-Saharan Africa. VF was disproportionately higher among younger adults, particularly those aged 18-25 years, where the rate reached 24.2%. Male patients also experienced higher VF rates (15.5%) compared to females (10.2%).

Site level differences were evident, with Nkwen Baptist Hospital recording a higher VF proportion (12.7%) than Bafoussam Baptist Hospital (9.0%) (Table 4).

Table 3: Demographic characteristics of the eligible cohort (n = 1,968).

Variable	Category	n	%
Sex	Male	554	28.2
	Female	1,414	71.8
Age category	18-25	128	6.5
	25-29	147	7.5
	30-34	227	11.5
	35-39	301	15.3
	40-44	306	15.5
	45-49	265	13.5
	50-54	212	10.8
	55+	382	19.4
Site	Nkwen Baptist Hospital	1,425	72.4
	Bafoussam Baptist Hospital	543	27.6

Table 4: Stratified virological failure (VF) rates by site, sex, and age group.

Stratum	Level	VF, n	Total, n	VF, %
Site	Bafoussam Baptist Hospital	49	543	9
	Nkwen Baptist Hospital	181	1,425	12.7
Sex	Male	86	554	15.5
	Female	144	1,414	10.2
Age group	18-25	31	128	24.2
	25-29	13	147	8.8
	30-34	32	227	14.1
	35-39	35	301	11.6
	40-44	25	306	8.2
	45-49	35	265	13.2
	50-54	27	212	12.7
	55+	32	382	8.4

Machine Learning Model Performance

Cross Validated Training Performance Table 5 reports 5-fold cross-validated accuracy and Cohen’s kappa on the training partition (post-upsampling), averaged across folds with 95% confidence intervals. Confidence intervals are wide, reflecting the small per fold sample size inherent to 5-fold cross-validation on this dataset.

Table 5: Cross validated (training) performance across candidate models.

Model	Accuracy (95% CI)
Logistic model	0.678 (0.558-0.797)
Random forest	0.700 (0.517-0.883)
Gradient boosting machine	0.703 (0.589-0.817)
SVM (radial basis function kernel)	0.702 (0.531-0.873)

Held Out Test Set Performance

Sensitivity is a critical measure in predictive modeling for HIV care, as it reflects the ability to correctly identify patients experiencing virological failure. A minimum sensitivity of $\geq 70\%$ is generally considered significant, while $\geq 80\%$ is preferred for programmatic use. All four algorithms were evaluated once on the held-out test partition. Discrimination and overall classification performance are summarized in Table 6 & Figure 3; all estimates are shown with 95% confidence intervals as exported by the platform. From the predictions above we will realize that gradient boosting machine achieved the

highest discrimination (AUC 0.79, 95% CI 0.786–0.793) of the four algorithms, with a classification metrics sensitivity/specificity split of 0.8/0.6 at the exported 0.5 threshold; random forest achieved higher sensitivity (0.854) but at a substantial cost to specificity (0.45). Discrimination for the logistic model (AUC 0.62) was only modestly better than chance and remains broadly consistent in magnitude with the pooled AUC (0.668) reported across machine learning models for HIV treatment outcomes in the systematic review by Kwarah et al, which this study already cites. Comparative ROC curves for all four models are shown in Figure 4.

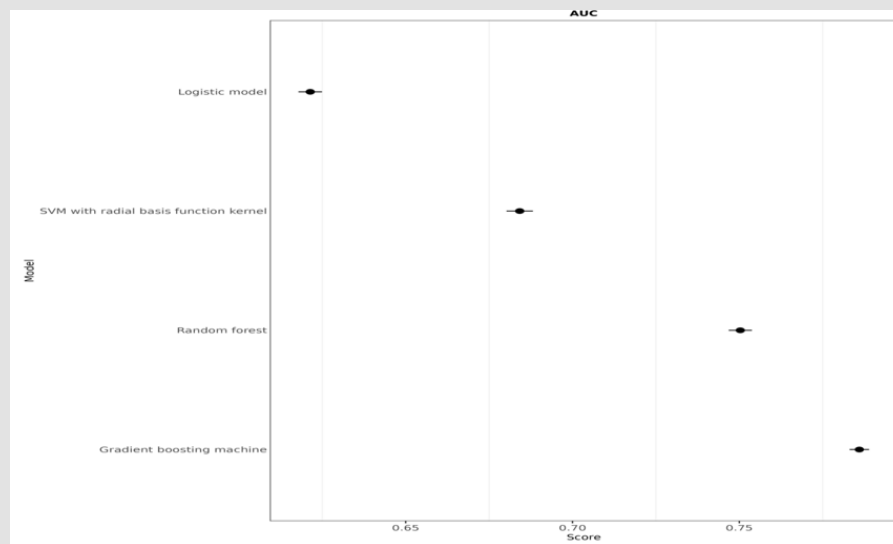


Figure 3: Model comparison.

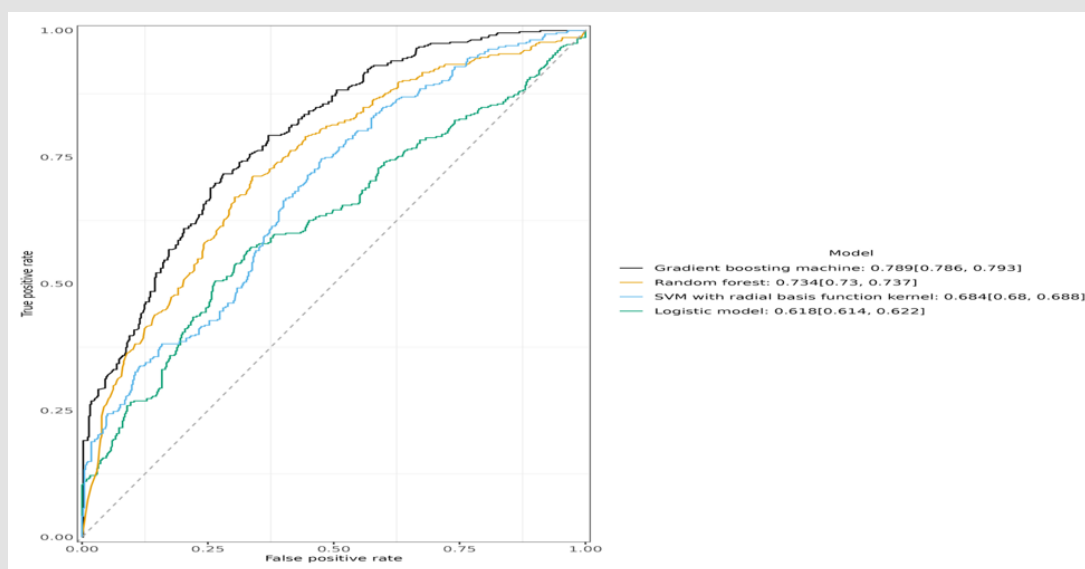


Figure 4: Receiver operating characteristic curves for the four candidate models on the held-out test set, with AUC and 95% CI shown in the legend.

Table 6: Model Comparisons.

Model	Accu	AUC	F1	logLoss	Sens	Spec
Gradient boosting machine	0.7	0.79	0.73	0.56	0.8	0.6
Logistic model	0.62	0.62	0.59	0.67	0.57	0.67
Random forest	0.64	0.73	0.7	1.16	0.84	0.45
SVM with rbf kernel	0.63	0.68	0.67	0.65	0.76	0.5

Feature Importance and Model Interpretation

SHAP Summary and Interpretation

SHAP based global importance analysis for the gradient boosting machine ranked body weight and age as overwhelmingly dominant contributors of prediction, well above the remaining predictors; this indicates that weight_kg patterns as well as age accounted for the majority of the predictive signal in the model, others such as site of enrolment, sex, and facility-based dispensation each contributed only marginally. Approximate relative importance read from Figure

5: weight.kg $\approx$ 85 (highest), Age $\approx$ 77, Sex_Male $\approx$ 0.5, dispensation.site_Facility $\approx$ 0.45 Site.Name_Nkwen.Baptist.Hospital $\approx$ 0.35 We equally see the confusion matrix for the gradient boosting machine, the best discriminating model. The confusion matrix shows how well our model separated patients who stayed suppressed from those who experienced virological failure. The model correctly predicted 260 true failures but missed 175, while it accurately predicted 347 suppressed patients and predicted 88 false alarms. Therefore the model predicted most failures and achieved about 70% overall accuracy as seen in Figure 6.

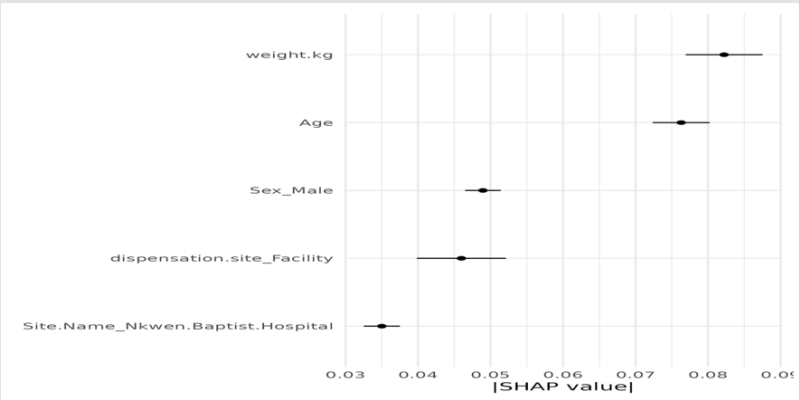


Figure 5: SHAP model interpretability.

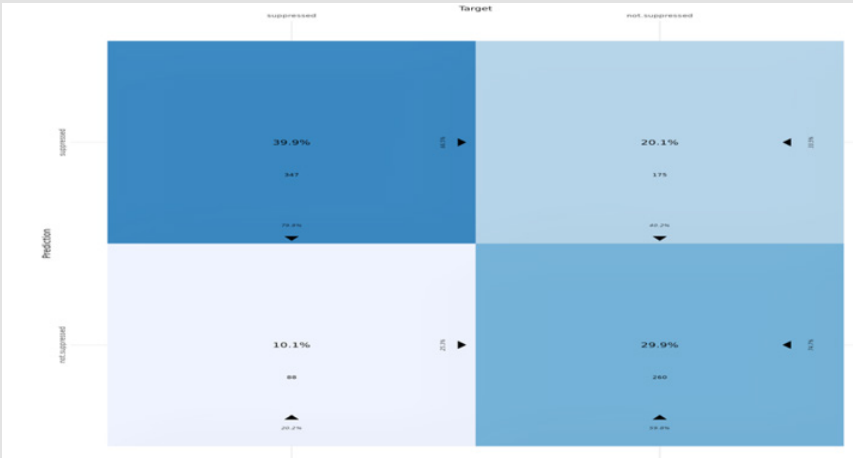


Figure 6: Confusion matrix for the gradient boosting machine model.

Discussion

This study shows that machine learning when applied to routinely collected clinical data, can effectively predict adult HIV patients at risk of virological failure with good accuracy in a Cameroonian ART setting and context. The gradient boosting model distinguished patients who went on to fail treatment from those who remained suppressed with strong discrimination, correctly flagging the large majority of true failures while keeping false alarms to a manageable minority. This is a meaningful result: it suggests that daily routine data already collected at ART centers, clinics or hospitals such as age, weight, sex, site, and dispensation pattern carry enough signal to support proactive, rather than purely reactive, identification of high-risk patients. Launching from the urgent backdrop that HIV remains one of the defining public health challenges of the twenty-first century with Cameroon reporting virological failure rates as high as 30% despite WHO's recommended ceiling of 10%. This finding directly addresses a critical gap in care, follow up and viral suppression strategies. The strength of this result is reinforced by its consistency with the broader literature: our model's discrimination compares favorably with the pooled performance of similar ML models for HIV treatment outcomes reported across other African settings, and it improves meaningfully on what a conventional logistic regression could extract from the same data, which performed only modestly above chance. Taken together, these results answer our research question directly: interpretable machine learning models, built on data readily available in Cameroonian ART clinics, can predict virological failure with clinically useful accuracy.

Beyond these findings, several secondary patterns were worth highlighting. Firstly, the tree-based ensemble methods (gradient boosting, random forest) consistently outperformed the linear logistic model, which mimicked a pattern seen elsewhere which is the relationships driving virological failure appear non-linear and interactive rather than additive, which is precisely the kind of structure ensemble methods are built to capture. In the second place we equally found a clear counter relationship between sensitivity and specificity across models, random forest somehow identified more true failures than gradient boosting, but at a high cost in false positives, jumping over half of suppressed patients as high risk. We chose the gradient boosting model because it strikes a better balance for clinics, too many false alerts would quickly cause clinical staff to distrust it, just as missed cases would.

And thirdly, body weight and age emerged as dominant predictors, overshadowing sex, dispensation site. This is a plausible finding because low weight is a recognized marker of advanced disease and poor treatment response but it was not the pattern we anticipated because we expected adherence related measures to be dominant but its absence from the final model reflects a deliberate methodological choice rather than a null finding: adherence proxy and CD4 count were removed during preprocessing because of strong collinearity

with other variables, so weight and age may be partly standing in for adherence and immunologic status rather than acting as fully independent drivers. Finally, our subgroup pattern showed an elevated risk among young adults and men which clearly aligns with well-documented vulnerabilities in HIV care globally, lending some external credibility to signals that were otherwise exploratory in this dataset. Our findings are subject to several limitations. Firstly, and most importantly, several variables which were central to Viral Failure risk such as CD4 count, clinical stage, BMI and profession had to be excluded rather than imputed because of very high missingness in the source registers. This would have almost certainly biased our model towards the variables that happened to be well recorded, rather than the variables most important to Viral Failure, and it means our reported performance should be read as a baseline on what richer and quality data could produce. And secondly, this was a retrospective, register based study, so our results inherit whatever inconsistencies that must have existed in routine clinical documentation; our adherence proxy in particular is a constructed stand in for true adherence behavior rather than a direct measurement of it and thirdly, the sample was drawn from two CBCHS affiliated hospitals in two regions, so generalizability to Cameroon's other regions and to non-CBCHS or more rural facilities cannot be assumed.

Finally fourth, model evaluation relied on a single held-out test split at one fixed threshold, rather than repeated or external validation, so our performance estimates while a reasonable first indication should be treated as preliminary rather than definitive and lastly the research based itself on one viral load outcome despite the recommendation from WHO defining a viral failure as two consecutive high viral load results, this limited our model yet gives us a picture, in the advent of future research, the use of two high viral load results will most certainly yield better result. Despite these constraints, several features of our design offer us some reassurance. We used a census of all eligible patients rather than a discrete sample, we equally applied a single standardized extraction checklist across both sites, and kept all preprocessing, imputation, balancing, encoding, strictly confined to the training partition to prevent bias from data leakage. Our use of SHAP alongside accuracy metrics also means that, unlike many prior ML based HIV models, ours does not ask clinicians to trust a black box; the model's reasoning is at least partly visible and clinically inspectable. Even with fewer variables than ideal, the model performed comparably to others developed in the region, showing that the missing data was not the only source of predictive signal in this population.

Despite these limitations, this study represents, to our knowledge, the first machine learning model developed and validated for predicting virological failure in the Cameroonian context, at a moment when comparable tools are already informing HIV care in Ethiopia, Ghana, and Nigeria. The practical implication is straightforward: a model of this kind could allow clinics to flag higher risk patients, young adults, men, and those with low body weight, for closer follow up or inten-

sified counselling shortly after treatment begins, rather than waiting for scheduled viral load results to reveal failure after the fact has already happened. This shifts Viral Failure management from a reactive point to a more proactive one, directly supporting progress toward the viral suppression goal of UNAIDS 95-95-95. Looking ahead, the clearest next steps are to validate this model prospectively and across additional sites, and to strengthen routine capture of CD4 count and clinical staging so that these clinically important variables can be brought back into future versions of the model without the missingness and collinearity problems encountered here. With these refinements, locally built and locally validated predictive tools of this kind could become a practical part of differentiated HIV care in Cameroon.

Conclusion

This study was set out to determine how accurately and interpretably machine learning could predict virological failure among adult HIV patients on ART in Cameroon, using routinely collected data at two Cameroon Baptist Convention Health Services facilities. Among 1,968 patients, virological failure prevalence was 11.7%, with markedly higher rates among young adults aged 18-25 years and among men. Of the four algorithms evaluated, the gradient boosting machine achieved the best overall discrimination (AUC 0.79) and the best-balanced sensitivity specificity ratio, thereby outperforming random forest, support vector machine, and logistic regression. SHAP based interpretation identified body weight and age as the dominant predictors, with site Nkwen Baptist Hospital, sex male, dispensation site despite a small probability, could offer clinicians a transparent account of the reasoning behind individual risk predictions rather than black-box reasoning. Taken together, these findings demonstrate that interpretable machine learning models built on routinely collected data can meaningfully predict HIV patients on ART at high risk of virological failure, even where key laboratory variables such as CD4 count, adherence proxies and WHO clinical stage are out of the picture.

This is, to our knowledge, the first model developed and validated in the Cameroonian context, and it fills a gap that has persisted even as comparable tools have advanced HIV care. The practical value of this work lies in its potential to shift virological failure detection from a reactive point depending on scheduled viral load testing to a more proactive one, in which higher risk patients are flagged for closer follow up, intensified adherence counselling, or earlier regimen review at or shortly after treatment initiation. Realizing this potential will require prospective validation across additional sites and regions, alongside sustained investment in more complete routine capture of CD4 count, clinical staging, and adherence data, so that future models can draw on the full range of clinically meaningful predictors. With these steps, locally developed and locally validated predictive tools of this kind can become a practical tool that can and could make a difference in HIV care in Cameroon, supporting continued progress toward the UNAIDS 95-95-95 targets.

Recommendations

Based on the findings of this study, the following recommendations are proposed for clinical practice, health system strengthening, and future research endeavors.

- 1. Prioritize Early Risk stratification.** ART clinics should consider incorporating age and body weight into routine risk assessment at or shortly after treatment initiation, given their dominant predictive contribution in this model. Patients under 25 years and those with low body weight at ART initiation warrant closer monitoring and more intensive adherence support from the onset, rather than waiting for scheduled viral load results.
- 2. Strengthen Adherence Support for Young Adults and Men.** The markedly elevated virological failure rates observed among 18-25-year-olds (24.2%) and male patients (15.5%) suggest these groups need specialized, targeted interventions such as peer support models, flexible clinic hours, and male friendly service delivery.
- 3. Pilot the Model as a Clinical Decision Support Tool.** Before a wider application, the gradient boosting model should first be tested in a few facilities as an added tool alongside routine viral load monitoring. Clinician feedback can then help refine how practical it is and whether its risk threshold is acceptable.
- 4. Invest in More Complete Routine Data Capture.** The very high missingness observed in CD4 count (96%), profession (93%), BMI (68%), and WHO clinical stage (43%) substantially limited the variables available for modeling. Strengthening data entry practices, register design and staff training at the point of care would allow these clinically important variables to be incorporated into future predictive models.
- 5. Consider Facility Level Quality Review Where Site Differences Persist.** The gap in VF rates between Nkwen (12.7%) and Bafoussam (9.0%) hospitals merits closer programmatic review to determine whether it reflects case-mix differences, adherence support infrastructure, or monitoring practices, so that facility specific improvements can be targeted appropriately.
- 6. Externally Validate the Model Across Additional Sites.** Future studies should test this model's performance in other Cameroonian regions and facility types, including rural and non-CBCHS sites, to establish how well it generalizes beyond the two urban and peri-urban hospitals studied here.
- 7. Re Incorporate Immunologic and Clinical Staging Variables.** As routine data quality improves, future models should test whether including CD4 count, WHO clinical stage, and BMI excluded here due to missingness improves predictive performance beyond what age and weight alone provide.

8. Conduct Prospective, Interventional Evaluation. A logical next step is a prospective study examining whether model guided flagging of high-risk patients, followed by targeted intervention, actually reduces virological failure rates compared to standard care moving beyond predictive accuracy to demonstrated clinical impact.

9. Investigate the Age and Sex Disparities Further. The concentration of risk among younger and male patients was exploratory in this analysis; dedicated studies examining the behavioral, social, and structural drivers behind these patterns would help translate this predictive signal into targeted, effective interventions.

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