International Journal of Community Medicine and Public Health
Rahayu D et al. Int J Community Med Public Health. 2025 Oct;12(10):4251-4259
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20252898

Original Research Article

Impact of baseline CD4 count on virologic suppression outcomes in people living with HIV on ART at Cibinong District Hospital

Dianita Rahayu[1]*, Ekowati Rahajeng[1], Anna Sunita[1], Iskari Ngadiarti[2]

[1]Department of Public Health, Universitas Respati Indonesia (URINDO), Jakarta, Indonesia
[2]Department of Nutrition, Poltekkes Kemenkes Jakarta II, Jakarta, Indonesia

Received: 15 August 2025
Accepted: 01 September 2025

***Correspondence:**
Dr. Dianita Rahayu,
E-mail: Dianita.drikavide@gmail.com

ABSTRACT

Background: Human immunodeficiency virus (HIV) remains a global health challenge, with viral suppression targets yet to be optimally achieved. A low baseline CD4 count at the initiation of therapy reflects immune system damage and increases the risk of viral suppression failure. This study aimed to assess the effect of baseline CD4 count on viral suppression failure among people living with HIV (PLHIV) receiving antiretroviral therapy (ART) at Cibinong District Hospital, Bogor Regency.

Methods: This case-control study utilized secondary data from electronic medical records. The study population included PLHIV who had been on ART for at least six months and had viral load (VL) results between November 2022 and March 2025 that met the inclusion and exclusion criteria. A total of 106 cases (VL>50 copies/ml) were selected through total sampling, while 212 controls (VL≤50 copies/ml) were randomly chosen. Multivariate logistic regression with a risk factor model was used for analysis.

Results: The findings revealed that 25.4% of PLHIV experienced viral suppression failure, the majority of whom had a baseline CD4 count <100 cells/mm³. Low baseline CD4 count significantly increased the risk of viral suppression failure (OR 4.5; 95% CI: 1.48-13.62) after adjusting for confounders (treatment duration, baseline BMI, CD4 change, baseline Hb, risk behaviors, TB history, and treatment adherence). An interaction was also identified between baseline CD4 count, nutritional status, and TB history, which further amplified the risk of viral suppression failure.

Conclusions: These findings underscore the importance of baseline CD4 testing at therapy initiation as a risk indicator. Early integration of HIV, TB, and nutrition services is essential to suppress viral replication and improve the quality of life of PLHIV.

Keywords: Baseline CD4, Nutritional status, Risk factors, TB-HIV

INTRODUCTION

Human immunodeficiency virus (HIV) infection attacks the immune system, particularly CD4 cells, which play a crucial role in defense against infections. The decline in CD4 count due to HIV infection weakens immune function and increases the risk of viral suppression failure.[1] Antiretroviral therapy (ART) remains the primary strategy to inhibit viral replication and achieve viral suppression, which in turn reduces morbidity, mortality, and HIV transmission.

HIV continues to pose a serious global health challenge. According to the World Health Organization (WHO), by the end of 2024 there were approximately 40.8 million people living with HIV (PLHIV), with 1.3 million new infections and 630,000 AIDS-related deaths. The UNAIDS 95-95-95 global targets, particularly the third indicator- achieving viral suppression in 95% of PLHIV on ART- have yet to be realized. Globally, 73% of PLHIV on ART have achieved viral suppression, 72% in Southeast Asia, while in Indonesia the proportion is only 46%, highlighting substantial challenges in achieving the 2030 target.[2,3]

Failure to achieve ART treatment targets is strongly influenced by the immunological status of PLHIV at treatment initiation, especially baseline CD4 count. A low CD4 count (<200 cells/mm³) indicates severe immunosuppression due to prolonged HIV infection, which is associated with poor immune response, delayed recovery, and a higher risk of viral suppression failure.[4] Several studies have demonstrated that low baseline CD4 count increases the likelihood of suppression failure. Derseh et al reported a 24.88-fold risk among PLHIV with CD4<250 cells/mm³, Meshesha et al found a 7.5-fold risk for CD4≤250 cells/mm³, Ahmed et al noted an odds ratio (OR) of 2.4 for CD4<200 cells/mm³, while Ayana et al reported more than a 2.3-fold increase for CD4<350 cells/mm³.[5-8] Desta et al observed a 1.33-fold higher risk for CD4<200 cells/mm³ compared with CD4≥500 cells/mm³, and Pérez-Molina et al documented an OR of 1.94 for low CD4 levels.[9,10]

In addition to baseline CD4 count, several other factors contribute to viral suppression failure in PLHIV. These include immunological factors such as a CD4 increase of <50 cells/mm³ in the first six months of therapy, which is linked to significantly higher risk, and clinical factors such as low BMI, advanced clinical stage, opportunistic infections, hemoglobin <12 mg/dl, and tuberculosis (TB) history.[8,11-15] Therapeutic factors such as ART regimen (e.g., nevirapine-based regimens carrying higher risk compared to efavirenz or dolutegravir), treatment adherence, and treatment duration also play a role.[13] Behavioral factors, including unprotected sex, injection drug use, and condomless anal sex, have been associated with increased risk of viral suppression failure.[15-17]

Cibinong District Hospital serves as a referral center for HIV care in Bogor Regency. As a type B teaching and regional referral hospital accredited with Paripurna status, it operates a multidisciplinary HIV/AIDS care team consisting of internists, pediatricians, neurologists, obstetricians, psychiatrists, psychologists, and trained HIV care staff. The HIV Care, Support, and Treatment (CST) Clinic was established in 2015, providing on-site CD4 and viral load testing integrated with electronic medical records (EMR). By the end of 2024, approximately 530 PLHIV were actively accessing ART at this facility. HIV program data are validated monthly in collaboration with the Bogor District Health Office to ensure accuracy in case numbers, drug availability, and viral load monitoring.

Program data from 2023-2024 revealed that 82.6% of PLHIV initiated ART with a baseline CD4 count <200 cells/mm³, and 40.9% of those who underwent viral load testing experienced suppression failure. To date, no study at Cibinong District Hospital has specifically examined the effect of baseline CD4 count on viral suppression failure using an etiological modeling approach that simultaneously controls for confounding factors.

This study aimed to analyze the effect of baseline CD4 count on viral suppression failure among PLHIV at Cibinong District Hospital, while considering clinical, therapeutic, and behavioral factors as potential confounders.

METHODS

This study employed a case-control design, conducted at the CST Clinic of Cibinong District Hospital, Bogor Regency, West Java, Indonesia. According to the Indonesian Ministry of Health, as of September 2024 there were 571,637 PLHIV in Indonesia, with 27,569 new cases and 27,349 AIDS-related deaths. Between January and September 2024, more than five million individuals underwent HIV testing, identifying 47,896 new cases. Java Island bears the highest burden, led by Jakarta, East Java, West Java, and Central Java, together with seven other provinces contributing 76% of national cases. In West Java, Bogor Regency ranks second after Bandung City in new HIV cases, with an increasing trend from 700 cases in 2023 to 814 cases in 2024. As a regional referral hospital, Cibinong District Hospital has provided HIV care for around 500 PLHIV since 2015, the majority of whom present with advanced clinical stages.

The study population included all PLHIV who had been on ART for at least six months. The target population was PLHIV on ART for ≥6 months with available viral load results, while the accessible population comprised PLHIV actively receiving ART at Cibinong District Hospital with viral load testing between November 2022 and March 2025. Cases were defined as PLHIV with viral load >50 copies/ml, and controls as those with viral load ≤50 copies/ml. The threshold of >50 copies/ml was applied to sensitively detect early suppression failure and provide a basis for early intervention.

Inclusion criteria were age ≥18 years and being on first-line ART regimens, while exclusion criteria included comorbidities (hypertension, diabetes mellitus, chronic kidney disease) and incomplete medical records. Sampling for cases was conducted using total sampling, while controls were selected randomly using Excel random tables. Sample size was calculated using Lemeshow's formula with 90% power and 95% confidence level, yielding 106 cases and 212 controls with a 1:2 ratio.

The primary independent variable was baseline CD4 count (≥200, 100 to <200, <100 cells/mm³). Confounding variables included clinical factors (CD4 change, baseline clinical stage, opportunistic infections, TB history, baseline BMI, baseline Hb), therapeutic factors (ART regimen, adherence, treatment duration), and behavioral factors (low, moderate, high risk). Data were extracted from EMR and standard laboratory tests. Interaction terms analyzed included baseline CD4 × TB history, given TB can accelerate CD4 decline through chronic inflammation and increased HIV replication, and baseline

CD4 × BMI, where malnutrition may impair CD4 regeneration and ART pharmacokinetics, thereby amplifying the risk of suppression failure in low CD4 patients.

Data collection was performed using a structured data extraction form from medical records, applying inclusion and exclusion criteria. All data were anonymized to maintain patient confidentiality. When EMR data were incomplete, additional verification was conducted through nursing summaries and ART clinic treatment notes, particularly for behavioral risk factors and adherence.

Statistical methods included descriptive analysis presented as frequencies and percentages. Multivariate analysis was conducted using logistic regression to estimate adjusted odds ratios (AOR) controlling for confounders. Interaction analysis assessed the relationship between baseline CD4 and TB history, as well as baseline CD4 and BMI, with significance set at $p<0.05$. Confounders were determined by evaluating changes in the CD4 odds ratio after removing candidate variables; variables were excluded from the model if the OR change was <10%. All analyses were performed using SPSS version 23, with $p<0.05$ considered statistically significant.

RESULTS

Figure 1 illustrates the sampling process. Among 613 PLHIV registered at the CST Clinic of Cibinong District Hospital who underwent viral load testing between November 2022 and March 2025, 156 (25.4%) experienced viral suppression failure and 457 (74.6%) were suppressed. A total of 248 PLHIV were excluded from the analysis due to ineligibility: aged <18 years (n=19), use of second-line regimens (n=2), presence of comorbidities (n=2), or incomplete medical records (n=225). Ultimately, 365 PLHIV met the eligibility criteria, with a 1:2 case-to-control ratio, resulting in 318 participants (106 cases and 212 controls) (Figure 1).

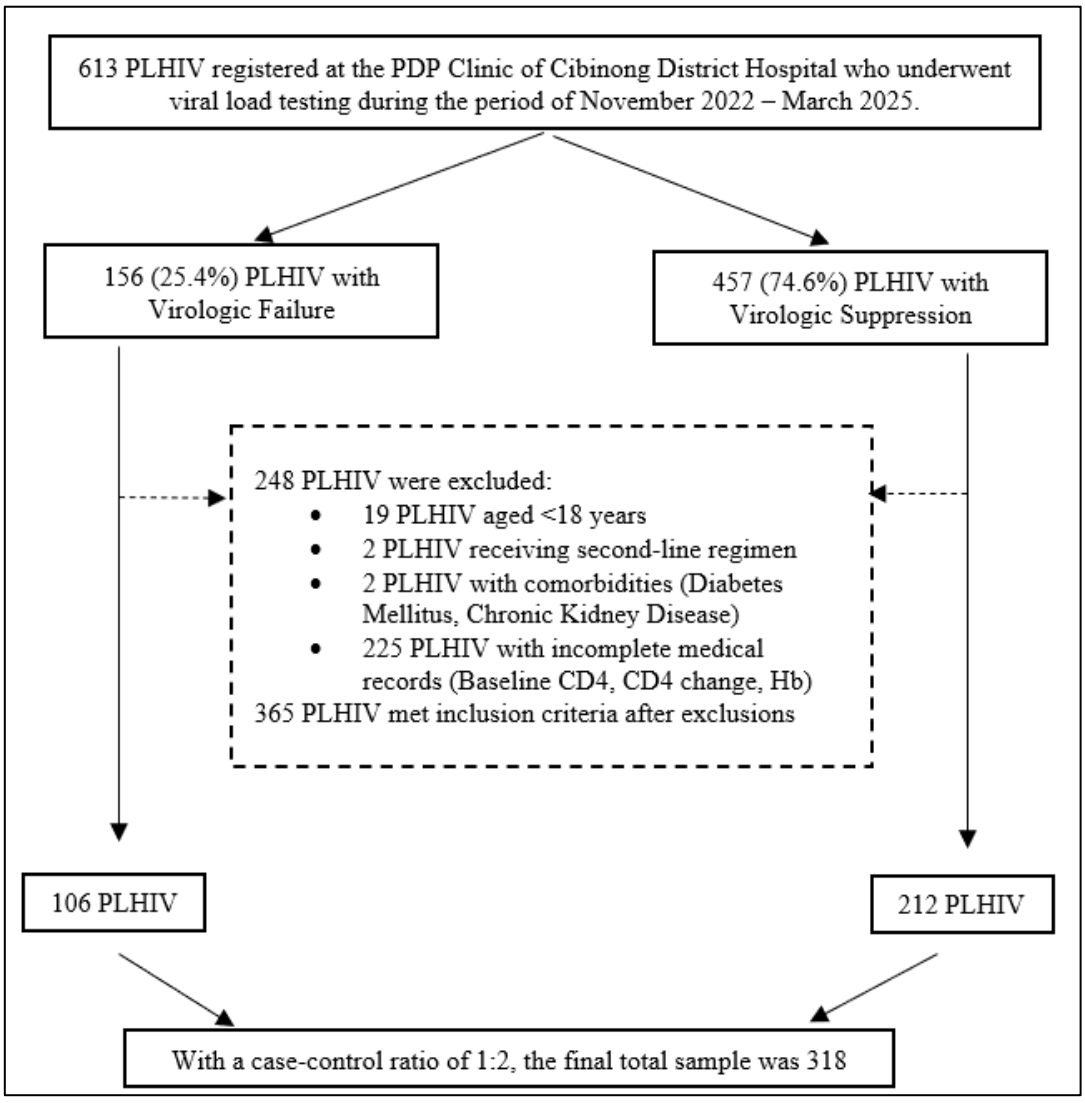

Figure 1: Sample selection flowchart.

Table 1 presents the characteristics of the 318 participants. Most respondents were aged ≥31 years (52.2%), male (78.3%), had secondary-level education (61.0%), worked as private employees (59.1%), and were unmarried (53.5%).

Clinical, therapeutic, and behavioral characteristics of the study population showed heterogeneous distributions across case and control groups. The highest proportion of viral suppression failure was observed among PLHIV with baseline CD4 <100 cells/mm^3 (55.7%), CD4 change <100 cells/mm^3 (36.8%), WHO clinical stage III-IV (81.1%), opportunistic infections (86.8%), and TB history (61.3%). In terms of nutritional status, underweight (BMI <18.5 kg/m^2) was more frequent among those with suppression failure (55.7%), while anemia (Hb<12 gm/dl) was also more common (49.1%). Regarding therapy, the majority of participants were on TLD (TDF+3TC+DTG) regimens (65.4%) across both groups. Good adherence was more prevalent among suppressed participants (71.2%), while poor adherence was observed in 20.8% of those with failure. Treatment duration ≥24 months was reported in about one-third of respondents in both groups. With respect to behavioral risk, medium-risk behaviors (MSM, female sex workers) predominated among suppression failure cases (77.4%), whereas low-risk behaviors (discordant couples, clients of sex workers) were more frequent among suppressed participants (41%) (Table 2).

Multivariate logistic regression revealed that PLHIV with baseline CD4 <100 cells/mm3 had a 4.48-fold higher risk of suppression failure compared to those with CD4≥200 cells/mm3 (AOR=4.48; 95% CI: 1.48-13.62), after controlling for confounders (treatment duration, baseline BMI, CD4 change, baseline Hb, behavioral risk, TB history, and adherence), as well as two interaction terms (baseline CD4 × BMI and baseline CD4 × TB history).

Table 1: Distribution of sociodemographic characteristics of PLHIV.

Variables	Viral suppression failure				Total	
	No (≤50 copies/ml)		Yes (>50 copies/ml)			
	N	%	N	%	N	%
Age (years)						
≥31	111	52.4	55	51.9	166	52.2
<31	101	47.6	51	48.1	152	47.8
Total	212	100	106	100	318	100
Sex						
Female	49	23.1	20	18.9	69	21.7
Male	163	76.9	86	81.1	249	78.3
Total	212	100	106	100	318	100
Education						
Diploma/bachelor/master	46	21.7	32	30.2	78	24.5
Senior high school	137	64.6	57	53.8	194	61.0
Junior high school	15	7.1	12	11.3	27	8.5
Elementary school	14	6.6	5	4.7	19	6.0
Total	212	100	106	100	318	100
Occupation						
Private sector employee	126	59.4	62	58.5	188	59.1
Entrepreneur	23	10.8	17	16.0	40	12.6
Civil servant	2	0.9	3	2.8	5	1.6
Army	0	0.0	1	0.9	1	0.3
Laborer	3	1.4	3	2.8	6	1.9
Retired	1	0.5	0	0.0	1	0.3
Housewife	17	8.0	9	8.5	26	8.2
College student	3	1.4	2	1.9	5	1.6
Unemployed	37	17.5	9	8.5	46	14.5
Total	212	100	106	100	318	100
Marital status						
Married	74	34.9	32	30.2	106	33.3
Widowed/divorced	25	11.8	17	16.0	42	13.2
Unmarried	113	53.3	57	53.8	170	53.5
Total	212	100	106	100	318	100

Table 2: Distribution of clinical, therapeutic, and behavioral characteristics.

Variables	Viral suppression failure				Total	
	No (≤50 copies/ml)		Yes (>50 copies/ml)			
	N	%	N	%	N	%
Baseline CD4 (cells/mm^3)						
≥200	80	37.7	22	20.8	102	32.1
100-<200	84	39.6	25	23.6	109	34.3
<100	49	22.6	59	55.7	107	33.6
Total	212	100	106	100	318	100
CD4 Change (cells/mm^3)						
≥100	140	66	67	63.2	207	65.1
50-<100	27	12.7	22	20.8	49	15.4
<50	45	21.2	17	16	62	19.5
Total	212	100	106	100	318	100
Clinical stage						
Clinical stage I-II	82	38.7	20	18.9	102	32.1
Clinical stage III-IV	130	61.3	86	81.1	216	67.9
Total	212	100	106	100	318	100
Opportunistic infections						
None	65	30.7	14	13.2	79	24.8
Present	147	69.3	92	86.8	239	75.2
Total	212	100	106	100	318	100
History of tuberculosis (TB)						
None	146	68.9	41	38.7	187	58.8
Present	66	31.1	65	61.3	131	41.2
Total	212	100	106	100	318	100
Baseline BMI (kg/m^2)						
18.5-25	118	55.7	43	40.6	161	50.6
>25	20	9.4	4	3.8	24	7.5
<18.5	74	34.9	59	55.7	133	41.8
Total	212	100	106	100	318	100
Baseline hemoglobin (Hb) (gm/dl)						
≥12	155	73.1	54	50.9	209	65.7
<12	57	26.9	52	49.1	109	34.3
Total	212	100	106	100	318	100
Drug regimen						
TLD (TDF+3TC+DTG)	137	64.6	71	67	208	65.4
TLE (TDF+3TC+EFV)	75	35.4	35	33	110	34.6
Total	212	100	106	100	318	100
Treatment adherence						
Good	151	71.2	60	56.6	211	66.4
Moderate	50	23.6	24	22.6	74	23.3
Poor	11	5.2	22	20.8	33	10.4
Total	212	100	106	100	318	100
Duration of treatment (months)						
≥24	66	31.1	31	29.2	97	30.5
<24	146	68.9	75	70.8	221	69.5
Total	212	100	106	100	318	100
Risk behavior						
Low risk (regular partner. sex worker clients)	87	41	22	20.8	109	34.3
Moderate risk (MSM. female sex workers)	120	56.6	82	77.4	202	63.5
High risk (people who inject drugs)	5	2.4	2	1.9	7	2.2
Total	212	100	106	100	318	100

Table 3: Full model.

Variables	P value	OR	95% CI
Baseline CD4 (cells/mm^3)			
≥200	0.001		
100-<200	0.333	0.553	0.167-10.834
<100	0.013	4.903	1.399-17.18
CD4 Change (cells/mm^3)			
≥100	0.034		
50-<100	0.017	2.818	1.203-6.603
<50s	0.653	0.832	0.374-1.852
Baseline clinical stage	0.803	0.889	0.353-2.24
Opportunistic infections	0.951	0.970	0.364-2.585
History of tuberculosis (TB)	0.001	7.587	2.185-26.351
Baseline BMI (kg/m^2)			
18.5-25	0.078		
>25	0.791	1.244	0.248-6.25
<18.5	0.031	0.181	0.038-0.857
Baseline hemoglobin (Hb)	0.010	2.365	1.233-4.537
Drug regimen	0.883	1.051	0.537-2.058
Treatment adherence			
Good	0.001		
Moderate	0.474	1.285	0.647-2.554
Poor	0.000	7.157	2.552-20.067
Duration of treatment	0.562	0.812	0.402-1.639
Risk behavior			
Low risk	0.000		
Moderate risk	0.000	4.203	2.098-8.422
High risk	0.786	1.364	0.145-12.811
Interaction: baseline CD4 × history of tuberculosis (TB)	0.010		
Baseline CD4 100-<200 cells/mm^3× history of tuberculosis (TB)	0.003	0.064	0.01-0.403
Baseline CD4<100 cells/mm^3× history of tuberculosis (TB)	0.039	0.183	0.037-0.915
Interaction: Baseline CD4× BMI	0.004		
Baseline CD4 100-<200 cells/mm^3× BMI>25 kg/m²	0.999	0.000	0-
Baseline CD4<100 cells/mm^3×BMI>25 kg/m²	0.742	0.613	0.033-11.277
Baseline CD4 100-<200 cells/mm^3× BMI<18.5 kg/m²	0.000	69.631	7.835-618.797
Baseline CD4<100 cells/mm^3× BMI<18.5 kg/m²	0.061	6.074	0.917-40.244

Table 4: Final multivariate model.

Variables	B	SE	Wald	df	Sig.	Exp(B)	95% CI for exp(B)	
							Lower	Upper
Baseline CD4 (cells/m^3)								
≥200			14.928	2	0.001			
100-<200	-0.637	0.592	1.158	1	0.282	0.529	0.166	1.687
<100	1.500	0.567	6.997	1	0.008	4.481	1.475	13.615
History of tuberculosis (TB)	1.963	0.601	10.653	1	0.001	7.122	2.191	23.151
Baseline BMI (kg/m^2)								
18.5-25			5.358	2	0.069			
>25	0.214	0.823	0.067	1	0.795	1.238	0.247	6.213
<18.5	-1.735	0.786	4.872	1	0.027	0.176	0.038	0.823
Duration of treatment	-0.217	0.337	0.416	1	0.519	0.805	0.416	1.557
CD4 Change (cells/m^3)								
≥100			6.790	2	0.034			
50-<100	1.022	0.431	5.630	1	0.018	2.778	1.195	6.460
<50s	-0.198	0.404	0.240	1	0.624	0.820	0.372	1.811

Continued.

Variables	B	SE	Wald	df	Sig.	Exp(B)	95% CI for exp(B)	
							Lower	Upper
Baseline hemoglobin (Hb)	0.851	0.330	6.660	1	0.010	2.342	1.227	4.469
Risk behavior								
Low risk			16.863	2	0.000			
Moderate risk	1.424	0.352	16.364	1	0.000	4.153	2.083	8.280
High risk	0.260	1.124	0.053	1	0.817	1.296	0.143	11.724
Treatment adherence								
Good			13.991	2	0.001			
Moderate	0.263	0.348	.572	1	0.450	1.301	0.658	2.572
Poor	1.965	0.526	13.987	1	0.000	7.137	2.548	19.992
Interaction: baseline CD4 × history of tuberculosis (TB			9.061	2	0.011			
Baseline CD4 100-<200 cells/mm^3× history of tuberculosis (TB)	-2.713	0.926	8.578	1	0.003	0.066	0.011	0.408
Baseline CD4<100 cells/mm^3× History of Tuberculosis (TB)	-1.663	0.808	4.233	1	0.040	0.190	0.039	0.924
Interaction: Baseline CD4 × BMI			15.433	4	.004			
Baseline CD4 100-<200 cells/mm^3× BMI>25 kg/m^2	-19.868	16258.787	0.000	1	.999	0.000	0.000	
Baseline CD4<100 cells/mm^3×BMI>25 kg/m^2	-0.455	1.475	0.095	1	0.758	0.634	0.035	11.431
Baseline CD4 100-<200 cells/mm^3× BMI<18.5 kg/m^2	4.258	1.109	14.753	1	0.000	70.679	8.047	620.816
Baseline CD4 < 100 cells/mm^3× BMI<18.5 kg/m^2	1.837	0.952	3.722	1	0.054	6.280	0.971	40.614
Constant	-3.046	0.572	28.359	1	0.000	0.048		

Interaction analysis showed significant effect modification. The baseline CD4 × TB history interaction indicated a lower risk of suppression failure among PLHIV with CD4 100-<200 cells/mm^3 and TB history (AOR=0.07; 95% CI: 0.01-0.41), and among those with CD4<100 cells/mm^3 and TB history (AOR=0.19; 95% CI: 0.04-0.92). Furthermore, a significant interaction between baseline CD4 × BMI was observed. The combination of CD4 100-<200 cells/mm^3 and BMI<18.5 kg/m^2 resulted in a markedly elevated risk of suppression failure (AOR=70.68; 95% CI: 8.05-620.82) (Table 3). The final model demonstrated that baseline CD4 count exerted a strong effect on virological suppression failure, with several confounders and effect-modifying interactions influencing the magnitude of risk.

DISCUSSION

This study demonstrated that baseline CD4 count, seven confounders (treatment duration, baseline BMI, CD4 change, baseline Hb, behavioral risk, TB history, and adherence), and two interactions (baseline CD4 × BMI and baseline CD4 × TB history) collectively influenced viral suppression failure in PLHIV (AOR=4.48; 95% CI: 1.48-13.62). A low baseline CD4 reflects severe immune damage, limiting the body's ability to control HIV replication despite ART.[4]

The findings of this study are consistent with those reported by Meshesha et al in Ethiopia, who demonstrated that patients with CD4 counts ≤250 cells/mm^3 had a 7.51-fold higher risk of virological suppression failure compared to those with CD4 counts >250 cells/mm^3. Other contributing factors included younger age (<35 years) and poor adherence, both of which substantially increased the risk of treatment failure.[6] Similarly, a study by Jaleta et al in Adama reinforced these results, showing that patients with CD4 counts <100 cells/mm^3 had a 3.29-fold increased risk, and those with CD4 counts between 100-200 cells/mm^3 had a 2.58-fold increased risk, compared to patients with CD4 counts ≥200 cells/mm^3. Moreover, suboptimal adherence was significantly associated with unsuppressed viral load.[23]

A meta-analysis conducted by Mou et al in China further highlighted that individuals with CD4 counts <200 cells/µl had a 7.04-fold greater risk of developing AIDS-related clinical events compared to those with higher CD4 levels. These findings underscore the pivotal role of baseline CD4 count as a prognostic indicator for ART success and patients' immunological status. Collectively, evidence from these studies strengthens the conclusion that lower baseline CD4 count not only increases the likelihood of virological suppression failure but also worsens long-term clinical outcomes.[24]

Importantly, this study highlights that suppression failure was not determined solely by baseline CD4, but also by its interaction with nutritional status and TB history. Our findings are consistent with Li et al, who reported that

higher baseline BMI was associated with improved long-term immunological recovery following ART initiation. Adequate nutrition supports immune cell function and homeostasis, as emphasized by Munteanu, who noted that optimal nutrition reduces inflammation and improves outcomes in immune-related conditions. Similarly, TB history was found to modify the effect of baseline CD4 on suppression failure. Chronic inflammation and immune activation associated with TB accelerate CD4 decline and impair immune function, thereby reducing ART effectiveness. Bruyn and Wilkinson emphasized the immunological interplay between CD4 and *Mycobacterium tuberculosis*, underscoring the importance of early ART initiation in HIV-TB co-infected patients.[18] Negash et al further demonstrated that TB co-infection delays immune recovery, especially in individuals initiating ART with very low CD4 counts, warranting close monitoring to optimize viral suppression and immune restoration.

The logistic regression model confirmed that baseline CD4 remained statistically significant after adjusting for confounders, supporting a causal relationship relevant for evidence-based clinical practice. These findings highlight the need for comprehensive HIV management that integrates immunological, nutritional, and co-morbidity considerations.

This study has several limitations, including reliance on secondary data, which limited the availability of certain potential confounders such as ART side effects, drug resistance, baseline viral load, psychological factors, and social support. Nevertheless, the use of multivariate modeling with interaction terms strengthens the validity of our findings and their applicability to clinical and public health interventions.

CONCLUSION

This study reinforces that low baseline CD4 count is an independent and consistent risk factor for viral suppression failure among PLHIV on ART, even after adjustment for confounders. The effect is further modified by nutritional status (baseline BMI) and TB history. These interactions highlight the importance of a comprehensive management approach that accounts for immunological status, nutritional support, and TB history. The findings support early screening, nutritional optimization, and integrated TB care as essential strategies to improve viral suppression outcomes in PLHIV.

ACKNOWLEDGEMENTS

The authors would like to express their gratitude to the director of Cibinong District Hospital and the staff for granting the opportunity to conduct this study, as well as to the HIV team of Cibinong District Hospital for their invaluable assistance in data collection and completion. Appreciation is also extended to the supervisors, professors, and lecturers of Universitas Respati Indonesia for their guidance and constructive feedback throughout the research process.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee of Universitas Respati Indonesia (Approval No. 206/SK.KEPK/UNRI/IV/2025) and received research permission from Cibinong District Hospital (Approval No. 800.2.4.2/2855-kepeg)

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Cite this article as: Rahayu D, Rahajeng E, Sunita A, Ngadiarti I. Impact of baseline CD4 count on virologic suppression outcomes in people living with HIV on ART at Cibinong District Hospital. Int J Community Med Public Health 2025;12:4251-9.