

Original Research Article

Factors associated with lack of viral suppression in human immunodeficiency virus-infected male patients treated with anti-retroviral therapy at Bach Mai hospital, Hanoi

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ABSTRACT

Background: Viral suppression following anti-retroviral therapy (ART) is one of the most effective factors determining human immunodeficiency virus (HIV) treatment success. However, certain numbers of HIV-infected patients do not experience viral suppression despite ART initiation, which ultimately might lead to treatment failure.

Method: A cross-sectional study was conducted including 194 male patients enrolled at the out-patient clinic, the Centre for Tropical Diseases, Bach Mai hospital, Vietnam. Data was analyzed using Stata 12.0.20 for Man-Whitney, Chi-square test/ Fisher's exact test, and multivariable logistic regression with statistically significant $p < 0.05$.

Results: The results show that being men who have sex with men (aOR=12.14, 95% CI: 1.48-99.49), having low CD4 T cell counts (aOR=269.58, 95% CI: 4.94-14721.27), living in rural areas (aOR=4.63, 95% CI: 1.04-20.57), and not having preventive tuberculosis treatment (aOR=9.92, 95% CI: 1.17-84.10), have increased odds of having detectable viral loads (VL). On the contrary, opportunistic infection was negatively associated with a lack of viral suppression (aOR=0.21, 95% CI: 0.05-0.96).

Conclusions: The results suggest that intervention programs should focus on patients living in rural areas, having low CD4 T cell counts, acquiring opportunistic infection, and MSM patients.

Keywords: HIV infection, Lack of viral suppression, Vietnam, CD4 T cell counts, Tuberculosis, Opportunistic infection

INTRODUCTION

Human immunodeficiency virus/ acquired immunodeficiency syndrome (HIV/AIDS) remains one of the leading causes of morbidity and mortality around the world. It was recently estimated that over 35.9 million adults are infected with HIV, however, only about 27.4 million (73%) are currently on ART, with 66% virally

suppressed.¹ The majority of newly infected patients are from low- and middle-income countries, such as Asia, Africa, and the Pacific.² Despite a global decline in HIV incidence, in Asia, the increase is disproportionate among different subpopulations, and the number of newly infected male cases remains significantly higher than females.³⁻⁵ In Asia, the proportion of patients who received ART increased dramatically from 17% in 2010

to 54% in 2018, nevertheless this number was still lower than the global figure (62%).^{3,4}

The HIV VL has been considered to be the most important marker to monitor disease progression and a VL >1,000 copies/ml is found to be associated with treatment failure and increased progression to AIDS.^{6,7} Most of the patients receiving ART treatment showed viral suppression after six months of therapy, which means the viral transmission was minimized.⁸ The guidelines by the Vietnamese ministry of health define viral suppression as undetectable VL (VL less than 20 copies/ml), associated with minimized risk of HIV transmission.⁹ However, the VL is analyzed only once per year, and up to 9.38-64.1% of patients still show detectable VLs, which eventually might lead to treatment failure, studying factors associated with detectable VL is crucial to prevent unsuppressed VL occurring in patients.¹⁰⁻¹²

Several factors associated with increased VL have been identified in different study settings including males, older age, having certain HLA genotypes, HIV clinical stage, low CD4 T cell counts (<50 cells/mm³), coinfection with sexually transmitted diseases, alcohol consumption, smoking, low body weight, prior tuberculosis, and ART duration.¹³⁻¹⁶ To better understand factors associated with lack of viral suppression in male patients in Vietnam, the demographic and clinical characteristics of patients enrolled at Bach Mai hospital, Hanoi, Vietnam, and the association with lack of viral suppression were assessed.

METHODS

The study was designed as a cross-sectional study including HIV-infected patients enrolled and treated with first line ART for at least one year at the outpatient's clinic at the Centre for Tropical Diseases, Bach Mai hospital, Hanoi, Vietnam from January to June 2019 (to have the record for CD4 T cell counts and HIV VL). Patients treated with second or third-line ART were excluded from the study. The patients were explained about the study objective and plan before signing the consent forms. Ethical permission was obtained from the institutional review board of Dinh Tien Hoang institute of medicine, Hanoi, Vietnam, under registration number IRB-007.

Clinical data, HIV VL, and factors associated with unsuppressed VL in HIV-infected patients receiving antiretroviral therapy were collected from the patients' medical records, including CD4 T cell counts, HIV VL, duration of ART, serum level of the liver enzymes alanine transaminase (ALT) and aspartate transaminase (AST), opportunistic infections (OIs), creatinine, hemoglobin, HIV/OI co-infection, clinical stage, sexually transmitted infection (STI), WHO clinical stage, hepatitis, tuberculosis, and preventive tuberculosis treatment.^{13,18} The demographic characteristics included gender, age,

body mass index (BMI), birthplace, living place, employment status, education, and alcohol consumption.

Data analysis

The studied outcome was a detectable VL (≥ 20 copies/ml). BMI is defined as weight in kg/squared height in meter (kg/m²). Weight status was categorized as underweight: BMI <18.5, healthy weight: BMI: 18.5-<25, and overweight or obesity: BMI ≥ 25 .¹⁷ The collected data were assembled using Epidata 3.1 (Epidata, "The Epidata Association" Odense, Denmark", <https://www.epidata.dk/index.htm>) and analyzed by Stata 12.0.20 (Stata Corp LLC, Texas, USA). Pearson's Chi-square/Fisher's exact test was used to compare proportions between detectable and undetectable VL groups with qualitative variables. Median with interquartile range (IQR) with Man-Whitney was used to present quantitative variables between two groups. Logistic regression was used to examine the associations' crude and adjusted odds ratios (OR) and 95% confidence intervals (95% CI).

RESULTS

A majority of patients were heterosexual men (60.8%). The age median was 33.6 years (IQR, 28-40 years). Most of them (79.4%) were in a normal BMI range. Using alcohol was reported by 71.6% of the participants. There were 37 patients (19%) having a history of tuberculosis infection, and 84 (43.3%) being positive for hepatitis. About 75.7% had at least one opportunistic infection. About 12.4% of the participants in this study had a detectable VL value (Table 1).

A difference between the two groups was observed about the percentage of alcohol consumption (69.4% in undetectable VL vs. 87.5% in detectable VL). Serum levels of clinical markers are shown in Table 2. There were a number of patients with elevated ALT and AST levels in both groups. The factors associated with the lack of viral suppression analyzed by univariate and multivariate models were presented in Table 3. Men who have sex with men (MSM) patients were more likely to have high VL compared to heterosexual patients (aOR=12.14, 95% CI: 1.48-99.49). In addition, participants living in rural areas were associated with high VL (aOR=4.63, 95% CI: 1.04-20.57). Not receiving tuberculosis preventive treatment was ten times as likely to have high VL (aOR=9.92, 95% CI: 1.17-84.10). CD4 cell count was inversely related to VL; compared to those with CD4 ≥ 350 cells/mm³, patients with a CD4 <100 cells/mm³ were 269 times as likely to have detectable VL (aOR=269.58, 95% CI: 4.94-14721.27), while patients with CD4 T cell counts between 250-350 cells/mm³ were associated with detectable VL (aOR=6.32, 95% CI: 1.25-31.06). On the contrary, opportunistic infection was negatively associated with detectable viral load (aOR=0.21, 95% CI: 0.05-0.96).

Table 1: Demographic and clinical characteristics of the HIV-infected study participants at Bach Mai hospital, Hanoi, Vietnam (n=194).

Variables	Total (%)
Gender	
Heterosexual	118 (60.8)
MSM	76 (39.2)
Living areas	
Urban	96 (49.5)
Rural	98 (50.5)
Age (in years)	Median: 33.6 years (IQR, 28-40 years)
Education	
≤Grade 5	10 (5.2)
6-9	38 (19.6)
10-12	57 (29.4)
>12	89 (45.9)
BMI (kg/m²)	
Underweight (<18.5)	22 (11.3)
Normal (18.5-24.9)	154 (79.4)
Overweight or obese (≥25)	18 (9.3)
Alcohol consumption	139 (71.6)
Tuberculosis preventive treatment	137 (70.6)
Hepatitis	84 (43.3)
Current opportunistic infections (OIs)	147 (75.8)
WHO clinical stage	
Stage I	149 (76.8)
Stage IV	1 (0.5)
CD4 T cell counts (cells/mm³)	
<100	3 (1.5)
100-<250	33 (17.0)
250-<350	28 (14.4)
≥350	130 (67.0)
VL value	
Detectable VL (≥20 copies/ml)	24 (12.4)
Undetectable VL	170 (87.6)

Table 2: Factors associated with lack of HIV viral suppression in HIV-infected male patients treated with ART analyzed by univariable and multivariable regression analysis.

Variables	Detectable VL		Undetectable VL		Crude OR (95% CI)	Adjusted OR (95% CI)
	N	%	N	%		
Gender						
MSM	13	17.1	63	82.9	2.01 (0.85-4.75)	12.14 (1.48-99.49)
Heterosexual men	11	9.3	107	90.7	1	1
Age group (in years)						
<25	5	25.0	15	75.0	1	1
25-<30	3	9.1	30	90.9	0.3 (0.06-1.43)	0.13 (0.01-1.38)
30-<35	4	13.8	25	86.2	0.48 (0.11-2.07)	0.24 (0.02-1.56)
35+	12	10.7	100	89.3	0.36 (0.11-1.17)	0.52 (0.06-4.44)
Living area						
Urban	8	8.3	88	91.7	1	1
Rural	16	16.3	82	83.7	2.15 (0.87-5.28)	4.63 (1.04-20.57)
BMI (kg/m²)						
Healthy weight (18.5-24.9)	2	9.1	20	90.9	1	1
Underweight (<18.5)	19	12.3	135	87.7	0.71 (0.15-3.28)	0.05 (0.002-1.10)
Overweight or obesity (≥25)	3	16.7	15	83.3	1.42 (0.38-5.37)	3.85 (0.43-34.80)

Continued.

Variables	Detectable VL		Undetectable VL		Crude OR (95% CI)	Adjusted OR (95% CI)
	N	%	N	%		
CD4 (cells/mm ³)						
≥350	13	10.0	117	90.0	1	1
250-<350	6	21.4	22	78.6	2.45 (0.84-7.15)	6.23 (1.25-31.06)
100-<250	4	12.1	29	87.9	1.24 (0.38-4.09)	0.61 (0.06-5.41)
<100	1	33.3	2	66.7	4.50 (0.38-53.09)	269.58 (4.94-14721.27)
AST levels						
High levels (>20)	3	12.5	21	87.5	1.01 (0.99-1.02)	1.68 (0.32-8.69)
Normal levels (10-20)	21	12.4	148	87.6	1	1
Opportunistic infection						
Yes	15	10.2	132	89.8	0.48 (0.19-1.18)	0.21 (0.05-0.96)
No	9	19.1	38	80.9	1	1
Sexually transmitted infection						
Yes	11	11.7	83	88.3	0.98 (0.97-1.00)	14.03 (0.72-272.78)
No	13	13.0	87	87.0	1	1
Tuberculosis preventive treatment						
No	3	5.3	54	94.7	0.42 (0.10-1.68)	9.92 (1.17-84.10)
Yes	14	10.2	123	89.8	1	1
Alcohol consumption						
Yes	21	15.1	118	84.9	1.21 (0.84-1.73)	1.55 (0.83-2.91)
No	7	12.7	48	87.3	1	1
Hepatitis						
Yes	9	10.7	75	89.3	0.76 (0.32-1.83)	0.58 (0.09-3.74)
No	15	13.6	95	86.4	1	1

Table 3: Factors associated with lack of HIV viral suppression in HIV-infected male patients treated with ART analyzed by univariable and multivariable regression analysis.

Variables	Crude OR (95%CI)	Adjusted OR (95%CI)
Gender		
Heterosexual men	1	1
MSM	2.01 (0.85-4.75)	12.14 (1.48-99.49)
Age group (in years)		
<25	1	1
25-<30	0.3 (0.06-1.43)	0.13 (0.01-1.38)
30-<35	0.48 (0.11-2.07)	0.24 (0.02-1.56)
35+	0.36 (0.11-1.17)	0.52 (0.06-4.44)
Living area		
Urban	1	1
Rural	2.15 (0.87-5.28)	4.63 (1.04-20.57)
BMI (kg/m²)		
Healthy weight (18.5-24.9)	1	1
Underweight (<18.5)	0.71 (0.15-3.28)	0.05 (0.002-1.10)
Overweight or Obese (≥25)	1.42 (0.38-5.37)	3.85 (0.43-34.80)
CD4 (cells/mm³)		
≥350	1	1
250-<350	2.45 (0.84-7.15)	6.23 (1.25-31.06)
100-<250	1.24 (0.38-4.09)	0.61 (0.06-5.41)
<100	4.50 (0.38-53.09)	269.58 (4.94-14721.27)
AST levels		
Normal levels (10-20)	1	1
High levels (>20)	1.01 (0.99-1.02)	1.68 (0.32-8.69)
Opportunistic infection		
No	1	1
Yes	0.48 (0.19-1.18)	0.21 (0.05-0.96)

Continued.

Variables	Crude OR (95%CI)	Adjusted OR (95%CI)
Sexually transmitted infection		
No	1	1
Yes	0.98 (0.97-1.00)	14.03 (0.72-272.78)
Tuberculosis preventive treatment		
Yes	1	1
No	0.42 (0.10-1.68)	9.92 (1.17-84.10)
Alcohol consumption		
No	1	1
Yes	1.21 (0.84-1.73)	1.55 (0.83-2.91)
Hepatitis		
No	1	1
Yes	0.76 (0.32-1.83)	0.58 (0.09-3.74)

DISCUSSION

The current study evaluated factors associated with detectable viral load in HIV-infected male patients treated with ART in Bach Mai hospital, Hanoi, Vietnam. Our analysis showed that being an MSM, having low CD4 T cell counts, not receiving tuberculosis preventive treatment and living in rural areas were found to be positively associated with lack of viral suppression, while acquiring at least one opportunistic infection was unlikely to be associated with detectable viral load.

HIV infected MSM were reported to have higher levels of viral load compared to heterosexual men with higher transmission odd ratio both before and after ART treatment.¹⁹⁻²¹ Higher HIV viral load was found in patients with low CD4 T cell counts. In this study, HIV infected MSM participants showed higher levels of CD4 T cell counts despite higher levels of viral replication. Another factor related to viral suppression was the early ART initiation.^{22,23} However, we could not access the time of ART initiation in HIV infected MSM, thus it is difficult to draw conclusions regarding the correlation between being MSM and having high levels of viral replication. Stirratt et al suggested that the increased censoring engagement in MSM participants might lead to condom less intercourse and thus facilitate sexual transmission from high viral load partners.²⁴

High HIV viral load was more likely to be found to be associated with markers related to HIV progression including low CD4, low body weight, and more advanced WHO clinical stages.¹³ AIDS-associated opportunistic infections at have been frequently found in patients with increased plasma HIV viremia levels.²⁵ However, in the current study, association between WHO clinical stage and elevated HIV viral load has not been observed, presumably due to limited patients at more advanced clinical stages. As acquiring opportunistic infection at WHO clinical stage I might not distort the immune function, including immune activation, increased viral replication might not be a direct consequence of opportunistic infection in the current participants. On the

contrary, an inverse correlation between opportunistic infection and VL were found. Most of the patients had been treated for opportunistic infection, which might be beneficial for patients in terms of viral replication.

Consistent with the previous finding, an association between lower CD4 T cell counts and elevated viral load levels was found.²⁶ Low CD4 T cell counts as the result of CD4 T cell depletion, especially memory T cells, was considered as one of the consequences of immune activation, reflected by increased T cell activation markers.²⁷⁻²⁹ Buzon et al hypothesized that elevation of HIV active replication might drive immune activation, thus leading to lower CD4 T cell counts.³⁰ CD4 T cell counts, and HIV viral load have been considered as the surrogate markers to monitor disease progression under ART treatment, and viral load has proved to be a better marker for monitoring the progression. Several studies have indicated that CD4 T cell counts could be more beneficial as predictor of HIV/AIDS progression compared to HIV viral load, suggesting that CD4 T cell counts are still an important marker for disease progression under certain circumstances.^{7,17,31}

Tuberculosis as the opportunistic infection has frequently been found in HIV infected patients in Vietnam and a link has been established between tuberculosis incidence and lack of virological suppression.³²⁻³⁷ However, it remains uncertain whether increased viral replication, combined with immune system deterioration, elevates the risk of tuberculosis acquisition, or if tuberculosis infection might facilitate viral replication in HIV-infected individuals. Goletti et al observed an increase of plasma viral load in patients with active tuberculosis (5-to 160-fold) and subsequent reduction of viral load as the tuberculosis treatment initiated, suggesting that controlling tuberculosis might be beneficial in suppressing HIV viral replication.³⁸ Pollack et al argued that the immune activation induced by mycobacterium tuberculosis may lead to further immune activation, weakening the immune response and thus, inability to suppress viral replication.¹³ Bulage et al suggested that tuberculosis treatment might have certain pharmacokinetics drug interactions with ART, thus favouring the ART treatment.³⁹ In line with

this, we found the association between not having tuberculosis preventive treatment and detectable viral load. Therefore, the tuberculosis preventive treatment might be beneficial in HIV infected patients in countries with high incidence of tuberculosis.

Living in rural areas was also found to be associated with lack of viral suppression. Higher levels of plasma viral load have been described as the consequence of increased drug resistance, especially in rural patients.⁴⁰ Limited transportation, shortage of healthcare services and cultural differences, and higher levels of social stigma in rural areas have been shown to account for the increased viral load.⁴¹ Additionally, Parker et al claimed that patients in rural areas face different barriers to care and treatment adherence, compared to persons in urban areas.⁴² Therefore, a more comprehensive intervention should be focused on patients living in rural areas in order to reduce treatment failure in these patients.

The current study investigated the factors associated with lack of viral suppression in HIV infected patients treated with ART at the outpatient clinic, Bach Mai hospital, Hanoi, Vietnam. There are several limitations in the study. Firstly, the patients were chosen in Bach Mai hospital, which might not represent the all-HIV infected men in Vietnam. Secondly, the study included possible associated factors, based on the established factors that have been found earlier, but other factors might be missing, which should be taken into consideration when interpreting the findings. Furthermore, the study was designed as a cross-sectional study so that only associated factors could be evaluated, risk factors and causal relationship cannot be analyzed. Finally, the analyses could be affected by the relatively low numbers of patients and other potential profound variables.

CONCLUSION

In the cohort of HIV infected patients treated with ART at Bach Mai hospital, Hanoi, Vietnam, being an MSM, having low CD4, living in rural areas and not having tuberculosis preventive treatment were found to be associated with increased odds of having a high VL, while having opportunistic infection might also be associated with viral suppression. The results suggest that further intervention programs should focus on HIV infected patients, especially MSM patients and patients living in rural areas.

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REFERENCES

1. The Global HIV/AIDS Epidemic. In: Services tUSDoHH, editor. 2021. Available at: <https://www.hiv.gov/>. Accessed on 14 November 2024.
2. Global HIV and AIDS statistics-Fact sheet. 2019. Available at: <https://www.unaids.org/en/resources/fact-sheet>. Accessed on 14 November 2024.
3. UNAIDS Data 2019. 2019. Available at: https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwjmehF-d3zAhUaat4KHZQkC3QQFnoECBQQAQ&url=https%3A%2F%2Fwww.unaids.org%2Fsites%2Fdefault%2Ffiles%2Fmedia_asset%2F2019-UNAIDS-data_en.pdf&usg=AOvVaw0PCW-o9HtZpIZRJ41Lqll7. Accessed on 14 November 2024
4. AIDSinfo. Updated. 2019. Available at: <https://aidsinfo.unaids.org/>. Accessed on 14 November 2024.
5. Number of new HIV infections. 2021. Available at: <https://www.who.int/data/gho/data/indicators/indicator-details/GHO/number-of-new-hiv-infections>. Accessed on 14 November 2024.
6. Mellors JW, Rinaldo CR Jr, Gupta P, White RM, Todd JA, Kingsley LA. Prognosis in HIV-1 infection predicted by the quantity of virus in plasma. *Science*. 1996;272(5265):1167-70.
7. Miller V, Phillips AN, Clotet B, Mocroft A, Ledergerber B, Kirk O, et al. Association of virus load, CD4 cell count, and treatment with clinical progression in human immunodeficiency virus-infected patients with very low CD4 cell counts. *J Infect Dis*. 2002;186(2):189-97.
8. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC, Kumarasamy N, et al. Prevention of HIV-1 infection with early antiretroviral therapy. *The New England journal of medicine*. 2011;365(6):493-505.
9. Control VNAfA. HIV/AIDS care and treatment guidelines. In: Vietnam MoH, editor. 2019.
10. Qiu T, Ding P, Fu G, Huan X, Xu X, Zhang Z, et al. Immunologic treatment failure among HIV-infected adult patients in Jiangsu province, China. *Sci Rep*. 2017;7:42381.
11. Samizi FG, Panga OD, Mulugu SS, Gitige CG, Mmbaga EJ. Rate and predictors of HIV virological failure among adults on first-line antiretroviral treatment in Dar Es Salaam, Tanzania. *J Infect Develop Countries*. 2021;15(6):853-60.
12. De La Hoz JM, Bolano L, Cardenas O, Gonzalez R, Sabbag J, Palacio L, et al. Characterization of

- treatment failure in HIV positive patients in the Colombian Caribbean region. *Colomb Med (Cali)*. 2014;45(4):162-7.
13. Pollack TM, Duong HT, Pham TT, Do CD, Colby D. Cigarette smoking is associated with high HIV viral load among adults presenting for antiretroviral therapy in Vietnam. *PLoS One*. 2017;12(3):e0173534.
14. Donnelly CA, Bartley LM, Ghani AC, Le Fevre AM, Kwong GP, Cowling BJ, et al. Gender difference in HIV-1 RNA viral loads. *HIV Med*. 2005;6(3):170-8.
15. Natural History Project Working Group for the Collaboration of Observational HIVEREiE. Factors associated with short-term changes in HIV viral load and CD4(+) cell count in antiretroviral-naïve individuals. *Aids*. 2014;28(9):1351-6.
16. Novitsky V, Gilbert P, Peter T, McLane MF, Gaolekwe S, Rybak N, et al. Association between virus-specific T-cell responses and plasma viral load in human immunodeficiency virus type 1 subtype C infection. *J Virol*. 2003;77(2):882-90.
17. Brown ER, Otieno P, Mbori-Ngacha DA, Farquhar C, Obimbo EM, Nduati R, et al. Comparison of CD4 cell count, viral load, and other markers for the prediction of mortality among HIV-1-infected Kenyan pregnant women. *J Infect Dis*. 2009;199(9):1292-300.
18. Joseph Davey D, Abrahams Z, Feinberg M, Prins M, Serrao C, Medeossi B, et al. Factors associated with recent unsuppressed viral load in HIV-1-infected patients in care on first-line antiretroviral therapy in South Africa. *Int J STD AIDS*. 2018;29(6):603-10.
19. Das M, Chu PL, Santos GM, Scheer S, Vittinghoff E, McFarland W, et al. Decreases in community viral load are accompanied by reductions in new HIV infections in San Francisco. *PLoS One*. 2010;5(6):e11068.
20. Ellen JM, Kapogiannis B, Fortenberry JD, Xu J, Willard N, Duval A, et al. HIV viral load levels and CD4+ cell counts of youth in 14 cities. *Aids*. 2014;28(8):1213-9.
21. Hollingsworth TD, Pilcher CD, Hecht FM, Deeks SG, Fraser C. High Transmissibility During Early HIV Infection Among Men Who Have Sex With Men-San Francisco, California. *J Infect Dis*. 2015;211(11):1757-60.
22. Yeghiazarian L, Cumberland WG, Yang OO. A stochastic multi-scale model of HIV-1 transmission for decision-making: application to a MSM population. *PLoS One*. 2013;8(11):e70578.
23. Dimitrov D, Wood D, Ulrich A, Swan DA, Adamson B, Lama JR, et al. Projected effectiveness of HIV detection during early infection and rapid ART initiation among MSM and transgender women in Peru: A modeling study. *Infect Dis Model*. 2019;4:73-82.
24. Stirratt MJ, Marks G, O'Daniels C, Cachay ER, Sullivan M, Mugavero MJ, et al. Characterising HIV transmission risk among US patients with HIV in care: a cross-sectional study of sexual risk behaviour among individuals with viral load above 1500 copies/mL. *Sex Transm Infect*. 2018;94(3):206-11.
25. Donovan RM, Bush CE, Markowitz NP, Baxa DM, Saravolatz LD. Changes in virus load markers during AIDS-associated opportunistic diseases in human immunodeficiency virus-infected persons. *J Infect Dis*. 1996;174(2):401-3.
26. Shoko C, Chikobvu D. A superiority of viral load over CD4 cell count when predicting mortality in HIV patients on therapy. *BMC Infect Dis*. 2019;19(1):169.
27. Hunt PW, Martin JN, Sinclair E, Bredt B, Hagos E, Lampiris H, et al. T cell activation is associated with lower CD4+ T cell gains in human immunodeficiency virus-infected patients with sustained viral suppression during antiretroviral therapy. *J Infect Dis*. 2003;187(10):1534-43.
28. Lederman MM, Funderburg NT, Sekaly RP, Klatt NR, Hunt PW. Residual immune dysregulation syndrome in treated HIV infection. *Adv Immunol*. 2013;119:51-83.
29. Lederman MM, Calabrese L, Funderburg NT, Clagett B, Medvik K, Bonilla H, et al. Immunologic failure despite suppressive antiretroviral therapy is related to activation and turnover of memory CD4 cells. *J Infect Dis*. 2011;204(8):1217-26.
30. Buzon MJ, Massanella M, Llibre JM, Esteve A, Dahl V, Puertas MC, et al. HIV-1 replication and immune dynamics are affected by raltegravir intensification of HAART-suppressed subjects. *Nat Med*. 2010;16(4):460-5.
31. Chadha S, Bhalla P, Jha AK, Gautam H, Saini S, Anuradha S, et al. Disease progression and antiretroviral therapy in newly seropositive HIV subjects in a tertiary care hospital in North India. *J Infect Develop Countries*. 2013;7(2):110-5.
32. Dang LVP, Nguyen QH, Ishizaki A, Larsson M, Vu NTP, Do Duy C, et al. Prevalence of Opportunistic Infections and Associated Factors in HIV-Infected Men Who Have Sex With Men on Antiretroviral Therapy in Bach Mai Hospital, Hanoi, Vietnam: A Case-Control Study. *Am J Mens Health*. 2020;14(3):1557988320926743.
33. Tran NB, Houben RM, Hoang TQ, Nguyen TN, Borgdorff MW, Cobelens FG. HIV and tuberculosis in Ho Chi Minh City, Vietnam, 1997-2002. *Emerg Infect Dis*. 2007;13(10):1463-9.
34. Komati S, Shaw PA, Stubbs N, Mathibedi MJ, Malan L, Sangweni P, et al. Tuberculosis risk factors and mortality for HIV-infected persons receiving antiretroviral therapy in South Africa. *Aids*. 2010;24(12):1849-55.
35. El-Khatib Z, Ekstrom AM, Ledwaba J, Mohapi L, Laher F, Karstaedt A, et al. Viremia and drug resistance among HIV-1 patients on antiretroviral treatment: a cross-sectional study in Soweto, South Africa. *Aids*. 2010;24(11):1679-87.
36. Ahoua L, Guenther G, Pinoges L, Anguzu P, Chaix ML, Le Tiec C, et al. Risk factors for virological failure and subtherapeutic antiretroviral drug

- concentrations in HIV-positive adults treated in rural North-Western Uganda. *BMC Infect Dis*. 2009;9:81.
37. Gupta A, Wood R, Kaplan R, Bekker LG, Lawn SD. Prevalent and incident tuberculosis are independent risk factors for mortality among patients accessing antiretroviral therapy in South Africa. *PLoS One*. 2013;8(2):e55824.
 38. Goletti D, Weissman D, Jackson RW, Graham NM, Vlahov D, Klein RS, et al. Effect of Mycobacterium tuberculosis on HIV replication. Role of immune activation. *J Immunol*. 1996;157(3):1271-8.
 39. Bulage L, Ssewanyana I, Nankabirwa V, Nsubuga F, Kihembo C, Pande G, et al. Factors Associated with Virological Non-suppression among HIV-Positive Patients on Antiretroviral Therapy in Uganda, August 2014-July 2015. *BMC Infect Dis*. 2017;17(1):326.
 40. Musiime V, Kayiwa J, Kiconco M, Tamale W, Alima H, Mugerwa H, et al. Response to antiretroviral therapy of HIV type 1-infected children in urban and rural settings of Uganda. *AIDS Res Hum Retroviruses*. 2012;28(12):1647-57.
 41. Etta EM, Mavhandu L, Manhaeve C, McGonigle K, Jackson P, Rekosh D, et al. High level of HIV-1 drug resistance mutations in patients with unsuppressed viral loads in rural northern South Africa. *AIDS Res Ther*. 2017;14(1):36.
 42. Parker RD, Mangine CM, Hendricks BM, Cima MJ, Mcie S, Sarwari A. Adherence to HIV Treatment and Care at a Rural Appalachian HIV Clinic. *J Assoc Nurses AIDS Care*. 2017;28(1):67-74.

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