

COMPARISON OF SERUM ALBUMIN LEVEL WITH CD4 COUNT AS A MARKER OF IMMUNOSUPPRESSION IN HIV/AIDS PATIENTS: A PROSPECTIVE FOLLOW-UP STUDY FROM A SOUTH INDIAN TERTIARY CARE CENTRE

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ABSTRACT

Background: Human immunodeficiency virus infection is associated with progressive immune suppression and systemic inflammation, which may influence nutritional and biochemical parameters such as serum albumin. Identifying simple surrogate markers that correlate with immune status may improve clinical monitoring, especially in resource-limited settings. The objective is to estimate serum albumin levels and CD4 counts, and to compare serum albumin levels with CD4 cell counts as a marker of immunosuppression, among HIV/AIDS patients attending the antiretroviral therapy (ART) centre of the study institution. **Materials and Methods:** An observational follow-up study was conducted over 18 months among adults living with HIV receiving tenofovir-lamivudine-efavirenz (TLE)-based antiretroviral therapy at a tertiary care centre in Kuppam, Andhra Pradesh. Ninety participants were enrolled by purposive sampling after written informed consent. Baseline demographic, clinical, and laboratory data were collected, followed by reassessment at three months. Hypoalbuminemia was defined as serum albumin <3.5 g/dL. Paired t-tests and Chi-square tests were used for statistical analysis, with p<0.05 considered significant. **Results:** The study included 90 participants, predominantly males (67.78%), with most individuals aged between 41 and 59 years. Hypoalbuminemia at baseline was observed in 65.56% of participants and reduced to 51.11% at three months. Mean haemoglobin increased from 11.97±1.67 g/dL to 12.28±1.70 g/dL (p<0.001), while mean CD4 count rose from 313.97±172.96 cells/mm³ to 367.27±178.31 cells/mm³ (p<0.001). Serum albumin levels showed a significant increase from 3.32±0.51 g/dL at baseline to 3.86±0.45 g/dL at three months (p<0.001). Total leukocyte count did not change significantly (p=0.376). A statistically significant association was observed between hypoalbuminemia and lower CD4 categories at both baseline (p<0.001) and three months (p<0.001). **Conclusion:** Serum albumin levels improved alongside immunological recovery in adults living with HIV on antiretroviral therapy and showed a significant association with CD4 cell count, supporting its role as a useful adjunct marker of immune status.

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection

continue to be among the world's biggest health problems, with serious medical, social, and economic ramifications. Progressive immunosuppression, the hallmark of HIV infection,

is mainly caused by loss of CD4⁺ T cells, which are essential for coordinating immunological responses; tracking the level of immunosuppression is therefore essential for monitoring disease course, directing antiretroviral therapy (ART), and forecasting patient outcomes.^[1,2]

CD4⁺ T-cell count has long been considered the gold standard for determining an HIV-positive person's immunological state, with a substantial correlation between declining CD4 levels and heightened vulnerability to opportunistic infections and disease progression. Nevertheless, CD4 testing necessitates specialised equipment, skilled personnel, and substantial financial resources, posing difficulties in many low- and middle-income countries where the burden of HIV/AIDS is disproportionately high. Finding alternative, affordable, and readily available biomarkers that can act as surrogate indicators of immunosuppression has therefore drawn increasing attention.^[3,4,5]

Serum albumin, a hepatically synthesised protein essential for regulating inflammatory reactions, carrying hormones and medications, and preserving oncotic pressure, is a potential solution. Hypoalbuminemia has long been recognised as a sign of chronic disease and poor nutritional status, and reduced serum albumin levels have been linked to advanced HIV/AIDS, opportunistic infections, and higher mortality; several studies suggest that serum albumin may indicate the level of immunosuppression and serve as a surrogate marker for CD4 count, especially in resource-limited settings.^[6,7,8]

Clinical and practical factors support comparing serum albumin and CD4 count as indicators of immunosuppression: CD4 count is a direct indicator of immune competence, whereas serum albumin offers an indirect representation of the host's nutritional and inflammatory status, both closely related to immunological function.^[9,10,11] Hepatic dysfunction, opportunistic infections, chronic inflammation, and protein-energy deficiency are possible causes of hypoalbuminemia in HIV patients, and since these conditions often coincide with severe immunosuppression, serum albumin may serve as a marker of CD4 decline. Unlike CD4 testing, which necessitates flow cytometry, albumin estimation can be performed with straightforward, widely accessible, and inexpensive biochemical tests, making it especially useful in remote and resource-constrained locations.^[12,13,14]

Using serum albumin as a surrogate measure has important implications: it could improve healthcare providers' capacity to track HIV progression in resource-limited settings, facilitate population-level monitoring, and enhance prognostication given the association of hypoalbuminemia with both immunosuppression and mortality risk. Measuring serum albumin and CD4 count together may offer complementary information capturing the nutritional and immunological aspects of disease progression.^[15,16] This study therefore aims to

establish the role of serum albumin level as a surrogate marker in HIV/AIDS patients as compared to CD4 counts.

Aim and Objectives

Among HIV/AIDS patients attending the ART Centre in PESIMSR, Kuppam:

1. To estimate serum albumin levels and CD4 counts.
2. To compare serum albumin levels with CD4 cell counts as a marker of immunosuppression in HIV/AIDS patients.

MATERIALS AND METHODS

This prospective follow-up study was conducted at a tertiary care teaching hospital attached to a medical college in Kuppam, Andhra Pradesh, with participants recruited from the antiretroviral therapy centre functioning under the Department of General Medicine. The study was conducted over 18 months, from April 2024 to September 2025. Patients aged more than 18 years with confirmed HIV infection or AIDS who were already receiving TLE-based highly active antiretroviral therapy were included. Patients were excluded if they had any pre-existing hepatobiliary disease, renal disease or chronic kidney disease, gastrointestinal disorders known to affect serum albumin levels, clinical evidence of shock, or a history of burns within the preceding 21 days.

The sample size of 90 was calculated based on a previously published prospective observational study, considering an expected prevalence of 38.3%, a precision of 10%, and a 95% confidence level. Purposive sampling was adopted: all consecutive patients attending the ART centre during the study period were screened for eligibility, and those fulfilling inclusion criteria were enrolled after written informed consent until the required sample size was achieved.

After eligibility assessment, a complete general physical examination and detailed systemic examination were performed for every participant. Baseline clinical assessment included vital parameters (pulse rate, blood pressure, respiratory rate, and temperature) and relevant clinical history. Laboratory investigations including serum albumin, CD4 cell count, haemoglobin, and total leukocyte count were performed at baseline and repeated after three months of follow-up. Hypoalbuminemia was defined as a serum albumin concentration of less than 3.5 g/dL; participants with levels below this threshold were classified as having hypoalbuminemia, while those with values ≥ 3.5 g/dL were considered to have normal serum albumin.

Ethical approval was obtained from the institutional human ethics committee prior to commencement, and written informed consent was obtained from all participants. Confidentiality was maintained using unique identification numbers, and participation was

entirely voluntary. Data were entered into a spreadsheet and analysed using STATA version 14. Categorical variables were expressed as frequencies and percentages, and continuous variables as mean±standard deviation. Student's t-test and Chi-square test were applied to assess associations between variables, and the paired t-test was used to compare mean serum albumin, haemoglobin, CD4 count, and total leukocyte count between baseline and three months. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 90 participants were enrolled. The age category distribution showed a predominance of participants aged 51–59 years (30.00%), followed by 41–49 years (22.22%) and 20–29 years

(20.00%); participants aged 30–39 years constituted 14.44%, those aged 60–69 years 12.22%, and only 1.11% were below 20 years. Males formed 67.78% of the population and females 32.22%. All participants (100.00%) were on TLE-based antiretroviral therapy. Smoking was reported by 25.56%, alcohol consumption by 20.00%, and a history of drug use by 13.33%. Diabetes mellitus was present in 5.56%, hypertension in 14.44%, asthma in 7.78%, tuberculosis history in 13.33%, and epilepsy in 2.22% of participants. None of the participants had hepatobiliary disease, renal disease/chronic kidney disease, gastrointestinal disease affecting albumin, clinical shock, or recent burns, indicating a clinically stable cohort at baseline free of major confounding comorbidities. These characteristics are summarised in [Table 1].

Table 1: Baseline demographic and clinical characteristics of the study population (n=90)

Variable	n	%
Age <20 years	1	1.11%
Age 20–29 years	18	20.00%
Age 30–39 years	13	14.44%
Age 41–49 years	20	22.22%
Age 51–59 years	27	30.00%
Age 60–69 years	11	12.22%
Male sex	61	67.78%
Female sex	29	32.22%
On TLE-based HAART	90	100.00%
Smoking	23	25.56%
Alcohol consumption	18	20.00%
History of drug use	12	13.33%
Diabetes mellitus	5	5.56%
Hypertension	13	14.44%
Asthma	7	7.78%
Tuberculosis history	12	13.33%
Epilepsy	2	2.22%
Hepatobiliary / renal / GI disease affecting albumin, shock, or recent burns	0	0.00%

System-wise clinical examination at baseline revealed abnormal findings in a minority of participants: cardiovascular examination was abnormal in 16.67%, respiratory system in 15.56%, per-abdomen examination in 13.33%, and central nervous system examination in 10.00%, with the remainder normal in each system. Baseline vital

parameters showed a mean pulse rate of 81.91±12.09 bpm, mean systolic blood pressure of 118.81±16.83 mmHg, mean diastolic blood pressure of 72.86±9.71 mmHg, mean respiratory rate of 17.69±2.88 per minute, and mean temperature of 37.07±0.57°C, all within physiologically stable ranges. These findings are summarised in [Table 2].

Table 2: Baseline system-wise clinical examination findings and vital parameters (n=90)

Parameter	Abnormal n (%)	Normal n (%)
Cardiovascular system	15 (16.67%)	75 (83.33%)
Respiratory system	14 (15.56%)	76 (84.44%)
Per-abdomen examination	12 (13.33%)	78 (86.67%)
Central nervous system	9 (10.00%)	81 (90.00%)
Vital parameter	Mean ± SD	Range
Pulse (bpm)	81.91 ± 12.09	55–108
Systolic BP (mmHg)	118.81 ± 16.83	90–158
Diastolic BP (mmHg)	72.86 ± 9.71	55–100
Respiratory rate (/min)	17.69 ± 2.88	12–24
Temperature (°C)	37.07 ± 0.57	36.0–38.7

At baseline, hypoalbuminemia was present in 65.56% of participants, reducing to 51.11% at three-month follow-up. Comparison of laboratory parameters between baseline and three months showed a statistically significant increase in mean

haemoglobin, from 11.97±1.67 g/dL to 12.28±1.70 g/dL (paired t=−5.012, p<0.001), and in mean CD4 count, from 313.97±172.96 cells/mm³ to 367.27±178.31 cells/mm³ (t=−9.127, p<0.001). Mean serum albumin increased significantly from

3.32±0.51 g/dL to 3.86±0.45 g/dL ($t=-9.91$, $p<0.001$). In contrast, mean total leukocyte count changed from 6591.89±1490.80 to

6676.93±1810.20 per mm³, a difference that was not statistically significant ($t=-0.890$, $p=0.376$). These comparisons are presented in [Table 3].

Table 3: Comparison of hypoalbuminemia prevalence and laboratory parameters between baseline and three months (n=90)

Parameter	Baseline	3 months	t-value	p-value
Hypoalbuminemia, n (%)	59 (65.56%)	46 (51.11%)	—	—
Haemoglobin, mean ± SD (g/dL)	11.97 ± 1.67	12.28 ± 1.70	-5.012	<0.001*
Total leukocyte count, mean ± SD (/mm ³)	6591.89 ± 1490.80	6676.93 ± 1810.20	-0.890	0.376
CD4 count, mean ± SD (cells/mm ³)	313.97 ± 172.96	367.27 ± 178.31	-9.127	<0.001*
Serum albumin, mean ± SD (g/dL)	3.32 ± 0.51	3.86 ± 0.45	-9.91	<0.001*

* $p<0.05$, paired t-test.

A statistically significant association was observed between hypoalbuminemia and CD4 category at baseline ($p<0.001$): among participants without hypoalbuminemia, 74.19% had CD4 ≥ 350 cells/mm³, whereas among those with hypoalbuminemia, only 10.17% had CD4 ≥ 350 cells/mm³ and the majority fell into the 100–199 (40.68%) and 200–349 (45.76%) categories. A

similar pattern persisted at three months ($p<0.001$): 77.27% of participants without hypoalbuminemia had CD4 ≥ 350 cells/mm³, compared with only 21.74% among those with hypoalbuminemia, most of whom fell into the 200–349 (52.17%) and 100–199 (19.57%) categories. These associations are detailed in [Table 4].

Table 4: Association between hypoalbuminemia and CD4 category at baseline and at three months (n=90)

Time point	Hypoalbuminemia	CD4 <100 n (%)	CD4 100–199 n (%)	CD4 200–349 / ≥ 350 n (%)	p-value
Baseline	No (n=31)	0 (0.00%)	1 (3.23%)	7 (22.58%) / 23 (74.19%)	<0.001*
Baseline	Yes (n=59)	2 (3.39%)	24 (40.68%)	27 (45.76%) / 6 (10.17%)	
3 months	No (n=44)	1 (2.27%)	1 (2.27%)	8 (18.18%) / 34 (77.27%)	<0.001*
3 months	Yes (n=46)	3 (6.52%)	9 (19.57%)	24 (52.17%) / 10 (21.74%)	

*Chi-square test, $p<0.05$.

DISCUSSION

The present study provides insight into the relationship between immunological status and nutritional markers among adults living with HIV receiving antiretroviral therapy. The demographic profile, dominated by middle-aged individuals and a higher proportion of males, aligns with epidemiological trends reported in many ART cohorts from resource-limited settings, reflecting improved survival following widespread ART availability and possible gender-related differences in healthcare-seeking behaviour and testing uptake. The relatively stable clinical profile of participants, with minimal confounding comorbidities, allowed a focused evaluation of immunological and biochemical outcomes without major interference from systemic illness.

The absence of hepatobiliary disease, chronic kidney disease, gastrointestinal disorders affecting albumin, and acute systemic conditions such as shock or burns in this cohort contrasts with historical pre-ART and early-ART era cohorts that often reported higher frequencies of opportunistic infections and advanced disease at presentation, reflecting improved early diagnosis and programmatic linkage to care in the current ART era.

The improvement in haemoglobin levels over follow-up is consistent with earlier literature demonstrating haematological recovery following effective antiretroviral therapy; anaemia has long been recognised as a common complication of HIV infection, driven by chronic inflammation, bone marrow suppression, and opportunistic infections, with viral suppression following ART initiation leading to gradual normalisation of haematological parameters.^[6] In contrast, the lack of statistically significant change in total leukocyte count highlights its limited sensitivity as a marker of immunological recovery, as leukocyte counts often remain within normal ranges despite substantial immunosuppression and may fluctuate due to transient infections, stress, or medication effects rather than reflecting true immune reconstitution.

The significant improvement in CD4 cell counts reinforces the effectiveness of ART in restoring immune competence; CD4 recovery occurs progressively following viral suppression, and early, sustained ART has been associated with robust CD4 gains and reduced immune activation.^[8] The relationship between serum albumin and immune recovery observed here deserves particular attention: although traditionally viewed as a marker of nutritional status, emerging evidence suggests albumin also reflects systemic inflammation and

immune dysfunction in HIV infection, and the significant rise in albumin paralleling improvement in CD4 counts supports the hypothesis that albumin serves as a surrogate marker of overall physiological recovery.

The high prevalence of hypoalbuminemia at baseline is consistent with previous studies reporting low albumin levels even among clinically stable individuals with HIV; hypoalbuminemia has been shown to predict adverse non-AIDS outcomes independent of CD4 count and viral load.^[17] The significant association between hypoalbuminemia and lower CD4 categories at baseline in this study reinforces the biological plausibility of this relationship and mirrors findings from Northern Uganda, where individuals with lower CD4 counts were significantly more likely to exhibit hypoalbuminemia.^[18] The persistence of this association at three months further underscores the interdependence of nutritional and immunological status, consistent with observations that serum albumin may lag behind CD4 recovery in some individuals, reflecting ongoing inflammatory processes despite immunological improvement.^[19]

These findings are consistent with a substantial body of literature directly comparing serum albumin with CD4 count as a marker of immunosuppression in people living with HIV, including studies from India and other resource-limited settings demonstrating that serum albumin correlates closely with CD4 category and disease severity.^[20,21,22,23] The consistency of these associations across diverse geographic regions, including India, Africa, and Southeast Asia, enhances their generalisability and suggests a universal biological relationship rather than a context-specific effect.^[24]

From a programmatic perspective, the use of serum albumin as an adjunct marker may be particularly valuable in resource-limited settings where frequent CD4 testing is constrained, consistent with updated WHO guidance advocating holistic HIV care models that integrate clinical, laboratory, and nutritional assessments.^[25] The absence of severe comorbidities in this cohort strengthens the internal validity of the observed associations, since liver disease, renal dysfunction, and gastrointestinal pathology are known to significantly confound albumin levels; by excluding such conditions, the present study offers a clearer interpretation of albumin as a marker linked primarily to HIV-related immune status. Advanced HIV disease has also been shown to adversely affect quality of life and mortality even in the combined antiretroviral therapy era, further underscoring the value of simple adjunct markers for early identification of at-risk patients.^[26] Neurocognitive and neurological findings were minimal in this cohort, consistent with reports of reduced prevalence of severe HIV-associated neurocognitive disorder in the ART era, further supporting the concept of HIV as a chronic, manageable condition when therapy is initiated and maintained.^[27]

Overall, the present study demonstrates that serum albumin closely parallels immunological recovery in adults living with HIV receiving ART. Improvements in haemoglobin, CD4 counts, and albumin levels reflect broader physiological stabilisation and reduced disease burden, and the observed associations suggest that serum albumin may serve as a practical, low-cost adjunct marker for monitoring disease status, particularly where advanced immunological testing is limited.

Limitations

This study has certain limitations. The relatively short follow-up period of three months limited the assessment of long-term trends and outcomes, and immune recovery may plateau or reverse over longer durations in the presence of poor adherence or emerging resistance. The single-centre design may restrict the generalisability of findings to broader populations, and serum albumin was assessed as a surrogate marker of nutritional status without concurrent detailed dietary assessment. Outcomes were not stratified by sex despite evidence that women may demonstrate different immunological recovery trajectories, representing an area for future research, and the complex bidirectional relationship between nutrition and immunity, whereby improved immunity may enhance nutritional status while adequate nutrition is also essential for optimal immune recovery, could not be fully disentangled within the study design.

CONCLUSION

This study demonstrates that adults living with HIV on TLE-based antiretroviral therapy showed significant improvements in key haematological, immunological, and nutritional parameters over a three-month follow-up period. Notable increases in haemoglobin levels, CD4 cell counts, and serum albumin concentrations indicate a positive response to ongoing therapy and clinical stability in the absence of major comorbid conditions that could confound these outcomes.

The significant association between hypoalbuminemia and lower CD4 categories at both baseline and follow-up highlights the close relationship between nutritional status and immune function in people living with HIV. Monitoring serum albumin alongside immunological markers may therefore provide additional clinical insight into patient status and recovery, supporting its role as a useful, low-cost adjunct parameter in routine HIV care, particularly in settings where advanced immunological testing is limited.

REFERENCES

1. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC, Kumarasamy N, et al. Prevention of HIV-1 Infection with Early Antiretroviral Therapy. *N Engl J Med*. 2011;365(6):493–505.

2. Brenner BG, Roger M, Routy JP, Moisi D, Ntemgwa M, Matte C, et al. High rates of forward transmission events after acute/early HIV-1 infection. *J Infect Dis.* 2007;195(7):951–9.
3. Gay C, Dibben O, Anderson JA, Stacey A, Mayo AJ, Norris PJ, et al. Cross-sectional detection of acute HIV infection: timing of transmission, inflammation and antiretroviral therapy. *PLoS One.* 2011;6(5).
4. Ananworanich J, Schuetz A, Vandergeeten C, Sereti I, de Souza M, Rerknimitr R, et al. Impact of multi-targeted antiretroviral treatment on gut T cell depletion and HIV reservoir seeding during acute HIV infection. *PLoS One.* 2012;7(3).
5. Finzi D, Blankson J, Siliciano JD, Margolick JB, Chadwick K, Pierson T, et al. Latent infection of CD4+ T cells provides a mechanism for lifelong persistence of HIV-1, even in patients on effective combination therapy. *Nat Med.* 1999;5(5):512–7.
6. Kaufmann GR, Cunningham P, Kelleher AD, Zaunders J, Carr A, Vizzard J, et al. Patterns of viral dynamics during primary human immunodeficiency virus type 1 infection. *J Infect Dis.* 1998;178(6):1812–5.
7. Robb ML, Eller LA, Kibuuka H, Rono K, Maganga L, Nitayaphan S, et al. Prospective study of acute HIV-1 infection in adults in East Africa and Thailand. *N Engl J Med.* 2016;374(22):2120.
8. Jain V, Hartogensis W, Bacchetti P, Hunt PW, Hatano H, Sinclair E, et al. Antiretroviral therapy initiated within 6 months of HIV infection is associated with lower T-cell activation and smaller HIV reservoir size. *J Infect Dis.* 2013;208(8):1202–11.
9. Wawer MJ, Gray RH, Sewankambo NK, Serwadda D, Li X, Laeyendecker O, et al. Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *J Infect Dis.* 2005;191(9):1403–9.
10. Powers KA, Ghani AC, Miller WC, Hoffman IF, Pettifor AE, Kamanga G, et al. The role of acute and early HIV infection in the spread of HIV and implications for transmission prevention strategies in Lilongwe, Malawi: a modelling study. *Lancet.* 2011;378(9787):256–68.
11. Hill AL, Rosenbloom DIS, Fu F, Nowak MA, Siliciano RF. Predicting the outcomes of treatment to eradicate the latent reservoir for HIV-1. *Proc Natl Acad Sci U S A.* 2014;111(37):13475–80.
12. Lefrère JJ, Roudot-Thoraval F, Mariotti M, Thauvin M, Lerable J, Salpêtrier J, et al. The risk of disease progression is determined during the first year of human immunodeficiency virus type 1 infection. *J Infect Dis.* 1998;177(6):1541–8.
13. Pilcher CD, Tien HC, Eron JJ, Vernazza PL, Leu SY, Stewart PW, et al. Brief but efficient: acute HIV infection and the sexual transmission of HIV. *J Infect Dis.* 2004;189(10):1785–92.
14. Lavreys L, Baeten JM, Chohan V, McClelland RS, Hassan WM, Richardson BA, et al. Higher set point plasma viral load and more-severe acute HIV type 1 (HIV-1) illness predict mortality among high-risk HIV-1-infected African women. *Clin Infect Dis.* 2006;42(9):1333–9.
15. Sáez-Cirion A, Bacchus C, Hocqueloux L, Avettand-Fenoel V, Girault I, Lecroux C, et al. Post-treatment HIV-1 controllers with a long-term virological remission after the interruption of early initiated antiretroviral therapy: ANRS VISCONTI study. *PLoS Pathog.* 2013;9(3).
16. Ananworanich J, Fletcher JLK, Pinyakorn S, van Griensven F, Vandergeeten C, Schuetz A, et al. A novel acute HIV infection staging system based on 4th generation immunoassay. *Retrovirology.* 2013;10(1).
17. Ronit A, Sharma S, Baker JV, Mngqibisa R, Delory T, Caldeira L, et al. Serum albumin as a prognostic marker for serious non-AIDS endpoints in the strategic timing of antiretroviral treatment (START) study. *J Infect Dis.* 2018;217(3):405–12.
18. Ahmed AA, Hassan HA, Mswelo VE, Abdi AA, Nixson O, Omar HMS, et al. Hypoalbuminemia in HIV-infected patients: its determinants and correlation with CD4 count in Northern Uganda. *AIDS Res Ther.* 2025;22(1).
19. Sharma S, Jamra Y, Hawaldar S, Meshram A. Study of serum albumin as surrogate marker of immune suppression in patients living with HIV and AIDS. *Int J Adv Med.* 2016;152–6.
20. Singh H, Mangla D, Singh J, Neki NS, et al. Role of serum albumin level compared to CD4 cell count as a marker of immunosuppression in HIV/AIDS patients. *Int J Curr Res Biol Med.* 2017;2(11):11–17.
21. Sunil Pralhadrao H, Kant C, Phepale K, Kumar Mali M. Role of serum albumin level compared to CD4+ cell count as a marker of immunosuppression in HIV infection. *Indian J Basic Appl Med Res.* 2016;5(5):495–502.
22. Govindani V, Hande HM. Serum albumin level compared to CD4+ count as a marker of immunosuppression in HIV/AIDS patients: an observational cross-sectional study from South India. *Research Square* [preprint]. 2025.
23. Balgi V, Nayak V, K SD. Study of serum albumin level in subjects with HIV infection, in relation to CD4 count, as a marker of immune suppression. *Int J Contemp Med Res.* 2019.
24. Dusingize JC, Hoover DR, Shi Q, Mutimura E, Kiefer E, Cohen M, et al. Association of serum albumin with markers of nutritional status among HIV-infected and uninfected Rwandan women. *PLoS One.* 2012;7(4).
25. Hirschall G, Harries AD, Easterbrook PJ, Doherty MC, Ball A. The next generation of the World Health Organization's global antiretroviral guidance. *J Int AIDS Soc.* 2013;16.
26. Portilla-Tamarit J, Reus S, Portilla I, Fuster Ruiz-de-Apodaca MJ, Portilla J. Impact of advanced HIV disease on quality of life and mortality in the era of combined antiretroviral treatment. *J Clin Med.* 2021;10(4):1–13.
27. Vastag Z, Fira-Mladinescu O, Rosca EC. HIV-associated neurocognitive disorder (HAND): obstacles to early neuropsychological diagnosis. *Int J Gen Med.* 2022;15:4079–90.