ORIGINAL ARTICLE

Study of Thyroid Function in Newly Diagnosed Human Immunodeficiency Virus Patients and Effect of Antiretroviral Therapy on Thyroid Function

Satyam Kumar[1], Poonam Gupta[2*], Ajeet Kumar Chaurasia[3], Manoj Kumar Mathur[4], Sharad Varma[5]

Received: 26 June 2025; *Accepted:* 06 December 2025

Abstract

Background: Thyroid dysfunction is a frequently overlooked yet clinically significant comorbidity in human immunodeficiency virus (HIV)-infected individuals. The introduction of antiretroviral therapy (ART) has improved life expectancy but has also been associated with metabolic and endocrine disturbances, including thyroid abnormalities. Thyroid dysfunctions such as subclinical hypothyroidism, sick euthyroid syndrome, and overt hypothyroidism are increasingly recognized in both treatment-naïve and ART-experienced patients. However, limited data are available on thyroid function at the time of HIV diagnosis and its evolution following ART initiation, especially in the Indian population.

Materials and methods: This prospective observational study was conducted at Moti Lal Nehru Medical College, Prayagraj, including 100 newly diagnosed HIV patients aged ≥18 years. Baseline free T3, free T4, TSH, and CD4 were measured prior and 3 months after the start of ART. Statistical analysis was performed using SPSS version 27.0, with paired *t*-tests, analysis of variance (ANOVA), and Pearson correlation test to assess the changes and associations between thyroid parameters and ART, CD4 count, and demographic variables.

Results: The study focused on measuring serum free T3, free T4, and TSH levels at baseline and after ART initiation and analyzing their relationship with immunological status as indicated by CD4 count. The study population consisted of 100 individuals with a mean age of 37.29 ± 13.01 years, predominantly male (70%). At baseline, 62% of patients were euthyroid, while the remaining 38% showed thyroid dysfunction, primarily subclinical hypothyroidism and subclinical hyperthyroidism. Following 3 months of ART, the prevalence of thyroid dysfunction increased: euthyroid patients decreased to 41%, and cases of subclinical hypothyroidism, clinical hypothyroidism, and subclinical hyperthyroidism rose noticeably. This shift in thyroid status distribution was statistically significant (Chi-squared test, $p = 0.028$), suggesting a potential impact of ART on thyroid physiology.

In terms of hormone levels, the study observed a statistically significant increase in mean TSH values (from 4.23 ± 4.13 µIU/mL to 7.50 ± 7.85 µIU/mL, $p < 0.001$) and a significant decrease in free T3 levels (from 2.92 ± 0.88 pg/mL to 2.45 ± 1.00 pg/mL, $p = 0.005$) post-ART. Free T4 levels did not show a significant change ($p = 0.337$). These results align with existing literature suggesting that ART may unmask or exacerbate subclinical thyroid dysfunction, possibly through immune reconstitution or direct effects on thyroid metabolism.

Correlation analysis demonstrated a significant negative association between CD4 count and TSH levels both before and after ART ($\rho = -0.28$ and -0.34, respectively), and a positive correlation between CD4 and both free T3 and free T4.

Conclusion: This study establishes that ART is associated with significant changes in thyroid hormone profiles, particularly an increase in TSH and a decline in free T3 levels, reflecting emerging thyroid dysfunction. The results underscore the importance of regular thyroid function monitoring in HIV patients, particularly after initiating ART, to facilitate early detection and management of evolving endocrine disturbances.

Journal of The Association of Physicians of India (2026): 10.59556/japi.74.1429

Introduction

Since its discovery in 1983, human immunodeficiency virus (HIV) has killed about 40.4 million people globally as of 2022. This figure is startling, and HIV might develop into a worldwide health emergency if unchecked. HIV belongs to the *Lentivirus* genus, Retroviridae family. The virus primarily attacks CD4+ T-lymphocyte helper cells, which results in severe immunological suppression and ongoing cell death. Numerous clinical symptoms result from this suppression, which also impairs the immune system. HIV eventually develops into AIDS if left untreated. At this point, opportunistic infections cause death because the immune system is unable to stop infections. HIV-1 and HIV-2 are the two primary forms of HIV. Numerous bodily fluids, including blood, amniotic fluid, breast milk, semen, rectal fluids, and vaginal fluids, can spread HIV. Sexual contact, pregnancy and childbirth, and fomites—such as needles or reusable medical equipment—can all spread HIV.[1]

By secreting thyroid hormones, the thyroid gland plays a crucial part in controlling growth, development, and metabolism. These hormones, triiodothyronine (T3) and thyroxine (T4), influence almost all organ systems and regulate a vast range of physiological functions. Thyroid disorders can have serious clinical repercussions that affect metabolism, mental clarity, cardiovascular health, and general quality of life. Significant changes in thyroid function and other aspects of the endocrine system can occur in the setting of chronic disorders like HIV infection. Comprehensive management of afflicted people requires an understanding of the interactions among HIV, antiretroviral medication (ART), and thyroid function.

Thyroid dysfunction in HIV-positive people is becoming more well recognized. These patients exhibit a wide range of thyroid abnormalities, from overt thyroid illness to subclinical hypothyroidism and euthyroid sick syndrome. Numerous theories have been put out to account for these changes, including autoimmune reactions, opportunistic thyroid infections, direct viral impacts on the thyroid gland, and the metabolic effects of long-term sickness.[2] Additionally, despite being life-saving, ART has been linked to the emergence of metabolic disorders, such as thyroid dysfunction.

Many HIV-positive individuals show symptoms of nonthyroidal illness syndrome before starting ART, which is typified by low serum T3 levels and normal or low thyroid-stimulating hormone (TSH) and T4 levels. Instead of being a primary thyroid

[1]Junior Resident; [2]Professor and Head; [3,4]Professor; [5]Senior Medical Officer, Department of Medicine, MLN Medical College, Prayagraj, Uttar Pradesh, India; *Corresponding Author

How to cite this article: Kumar S, Gupta P, Chaurasia AK, *et al.* Study of Thyroid Function in Newly Diagnosed Human Immunodeficiency Virus Patients and Effect of Antiretroviral Therapy on Thyroid Function. J Assoc Physicians India 2026;74(3):84–89.

disease, this disorder is an adaptive reaction to a systemic ailment. After starting ART, a complicated series of changes could take place. Hashimoto's thyroiditis and Graves' disease are two autoimmune thyroid conditions that might be concealed by immunological reconstitution inflammatory syndrome (IRIS).[3] Furthermore, direct thyroidal effects have been linked to some antiretroviral medications, while the precise pathways are still being studied. Geographic location, stage of HIV infection, ART regimen, and the presence of opportunistic infections are some of the factors that affect the prevalence rates of thyroid dysfunction in HIV-positive populations, according to epidemiological research.

Aims and Objectives

- To measure serum free T3, serum free T4, and serum TSH levels in newly diagnosed HIV patients to be started on ART.
- To study the association between thyroid hormone levels and ART regimen in newly diagnosed HIV patients.
- To study thyroid function after 3 months of initiating ART.

Materials and Methods

Before the study, proper ethical clearance was obtained from the institute.

Place of Study

This prospective observational study was conducted at SRN Hospital, MLN Medical College, Prayagraj.

Duration of Study

The study was carried out over a 12-month period from 1st March 2024 to 1st March 2025.

Type of Study

Prospective observational study.

Study Population

Newly diagnosed HIV patients (aged ≥18 years) enrolled from OPD and IPD of the Department of Medicine and ART Center.

Sample Size

Hundred.

Sample Size Calculation

Based on the study by Dev et al. (2015), reporting prevalence rates of thyroid dysfunction between 30 and 75%. For conservative estimation, a prevalence (p) of 30% (0.30) was selected as the expected proportion.

For a proportion-based study design, the following formula was used:

$n = (Z^2 \times p \times (1 - p))/d^2$

Where:

n = required sample size

Z = Z-score corresponding to the desired confidence level (1.96 for 95%)

p = expected prevalence/proportion (0.30)

d = desired margin of error or precision (0.10 i.e., 10%)

$n = (1.96)^2 \times 0.30 \times (1 - 0.30) / (0.10)^2$

$n = 3.8416 \times 0.30 \times 0.70 / 0.01$

$n = 0.806736 / 0.01$

$n = 80.67 \approx 81$ patients

To account for a potential dropout rate of 20%, the sample size was adjusted:

Final sample size = $81 \times 1.20 = 97.2 \approx 100$ patients

Inclusion Criteria

- Newly diagnosed HIV cases aged ≥18 years.

Exclusion Criteria

- Patients on medications such as amiodarone, lithium, and immunosuppressants.
- Pregnant and lactating females.

Study Procedure

After obtaining informed written consent from each participant, newly diagnosed HIV-positive patients were systematically enrolled into the study. A detailed history was recorded, focusing on the presence of any known thyroid disorder, current pregnancy or lactation status, and any prior exposure to antiretroviral therapy (ART). This was followed by a comprehensive physical examination and a full general and systemic examination to assess the clinical status of the patient.

Relevant laboratory investigations were conducted, including thyroid function tests (free T3, free T4, and serum TSH), HIV-1 and HIV-2 confirmation, CD4 lymphocyte count, complete blood count parameters (hemoglobin, total leukocyte count, platelet count, MCH, MCHC, MCV), liver function tests (SGOT/AST, SGPT/ALT), and renal function tests (serum urea and creatinine). All clinical and biochemical findings were meticulously documented to evaluate the baseline health profile of the participants and to facilitate follow-up analysis of thyroid function after the initiation of ART.

Statistical Analysis

Statistical analysis was performed using SPSS software version 27.0. Descriptive statistics, including mean and standard deviation, were applied to summarize demographic variables, hematological, biochemical, and immunological parameters. Paired t-tests were used to compare thyroid hormone levels (free T3, free T4, and TSH) before and after 3 months of ART, assessing the significance of observed changes. The Chi-squared test was utilized to evaluate changes in the categorical distribution of thyroid status (euthyroid, hypothyroid, and hyperthyroid) pre- and post-ART. Spearman's rank correlation coefficient was employed to analyze the association between CD4 count and thyroid parameters, both at baseline and post-ART, including delta (Δ) changes. Lastly, multivariate logistic regression analysis was conducted to identify independent predictors of thyroid dysfunction post-ART. A p-value of <0.05 was considered statistically significant.

Results

The age distribution of the study participants (n = 100) shows that the majority belonged to the younger age-groups, with the highest proportion (34%) aged between 20–29 years (n = 34), followed by 25% in the 30–39 years category (n = 25). Participants aged 40–49 years constituted 19% (n = 19), while those in the 50–59 years and 60–70 years comprised 11% (n = 11) and 8% (n = 8), respectively. Only 3 participants (3%) were below 20 years of age. The overall mean age of the cohort was 37.29 years with a standard deviation of ±13.01, indicating a fairly wide age spread and a predominant representation of individuals in early to mid-adulthood, as shown in Table 1 and its graphical representation in Figure 1.

The sex distribution of the study population (n = 100) reveals a male predominance, with 70% of the participants (n = 70) being male and 30% (n = 30) being female, as shown in Table 2 and its graphical representation as a pie chart in Figure 2.

The comparison of thyroid hormone levels before and after 3 months of ART reveals significant changes, particularly in TSH and free T3 values. The mean free T3 level declined from 2.92 ± 0.88 pg/mL at baseline to 2.45 ± 1.00 pg/mL post-ART, a statistically significant decrease (t = 2.877, p = 0.005), indicating a potential suppression of triiodothyronine levels following ART initiation. Free T4 levels, however, showed only a mild, statistically nonsignificant reduction from 1.19 ± 0.37 ng/mL to 1.10 ± 0.38 ng/mL (t = 0.965, p = 0.337), suggesting relatively stable thyroxine levels. In contrast, TSH levels demonstrated a marked increase from 4.23 ± 4.13 µIU/mL at baseline to 7.50 ± 7.85 µIU/mL post-ART, a highly significant change (t = –6.518, p < 0.001), indicating a potential shift toward hypothyroid status or subclinical thyroid dysfunction posttreatment. These results underscore the need for close thyroid function monitoring in patients undergoing ART, as shown in Table 3 and its graphical representation in Figure 3.

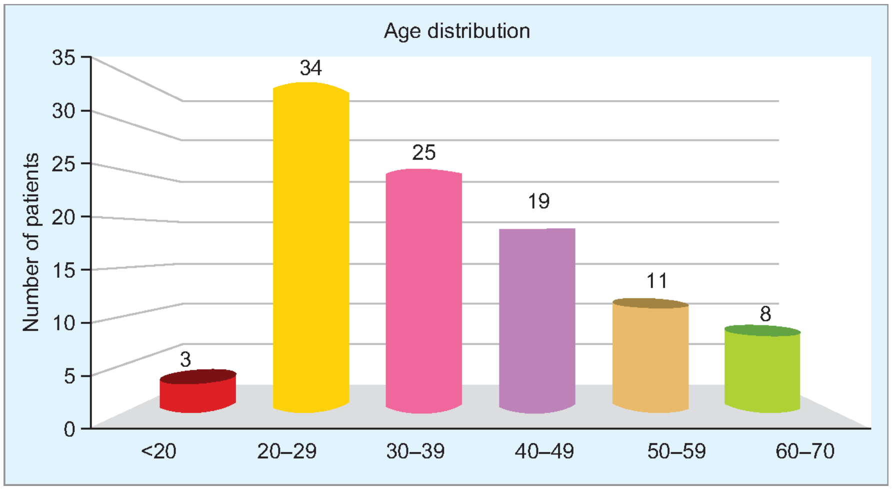


Fig. 1: Graphical representations of age distribution of enrolled patients

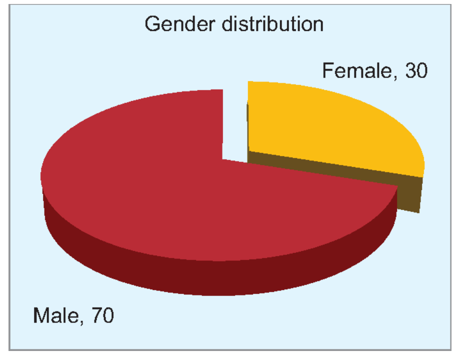


Fig. 2: Graphical representations of gender-wise distribution of enrolled patients

Table 1: Age-wise distribution of participants

Age distribution	*N*	*%*
<20	3	3.00%
20–29	34	34.00%
30–39	25	25.00%
40–49	19	19.00%
50–59	11	11.00%
60–70	8	8.00%
Mean ± SD	37.29 ± 13.01	

Table 2: Gender-wise distribution of enrolled patients

Gender distribution	*N*	*%*
Female	30	30.00%
Male	70	70.00%

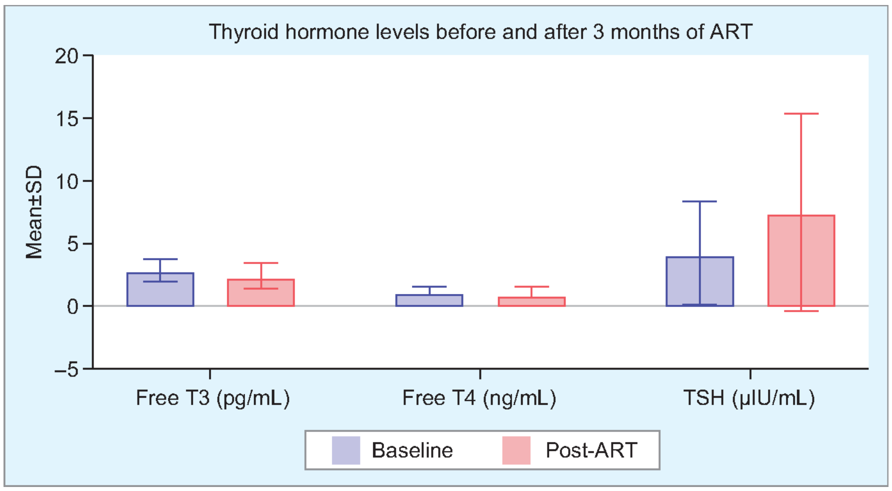


Fig. 3: Graphical representations of thyroid hormone levels before and after 3 months of ART

The distribution of patients according to thyroid status before and after ART demonstrates a statistically significant shift ($\chi^2 = 9.10$, $p = 0.028$), indicating that ART has an impact on thyroid function. At baseline, the majority of participants (62.0%) were euthyroid, but this proportion declined significantly to 41.0% post-ART. Conversely, there was a rise in all categories of thyroid dysfunction after therapy: subclinical hypothyroidism increased from 11.0 to 17.0%, clinical hypothyroidism rose from 14.0 to 19.0%, and subclinical hyperthyroidism showed the most pronounced increase from 13.0 to 23.0%, as shown in Table 4 and its graphical representation in Figure 4. These findings suggest that ART may unmask or induce thyroid dysfunction in a subset of patients, with a trend toward both hypothyroid and hyperthyroid changes.

At baseline, CD4 counts had a moderate negative correlation with TSH ($\rho = -0.28$, $p = 0.004$), indicating that lower immune status was associated with higher TSH levels. In contrast, free T3 and free T4 levels were positively correlated with baseline CD4 counts ($\rho = +0.35$ and $+0.31$, respectively, both $p \leq 0.002$), suggesting better thyroid hormone levels in individuals with higher immune function.

Following ART, these trends became more pronounced. The negative correlation between CD4 and TSH strengthened ($\rho = -0.34$, $p = 0.001$), while the positive correlation of CD4 with free T3 and free T4 also intensified ($\rho = +0.41$ and $+0.36$, respectively, both $p \leq 0.001$), reflecting improved thyroid function with immune restoration.

When examining the delta (Δ) values—representing changes from pre- to post-ART—a statistically significant inverse relationship was observed between ΔCD4 and ΔTSH ($\rho = -0.30$, $p = 0.003$), implying that increases in CD4 counts were linked to reductions in TSH levels. Likewise, positive correlations were noted between ΔCD4 and Δ free T3 ($\rho = +0.33$, $p = 0.002$) and Δ free T4 ($\rho = +0.29$, $p = 0.005$), as shown in Table 5 and their graphical representation in Figures 5A to C, indicating that immune recovery was associated with parallel improvements in thyroid hormone levels.

The multivariate logistic regression model evaluating predictors of thyroid dysfunction after ART among 100 patients did not identify any statistically significant independent variables. The intercept had a coefficient of −0.180 ($p = 0.887$), indicating no baseline risk in the absence of predictors. Age had a negative coefficient (−0.010, $p = 0.574$), suggesting a minimal and nonsignificant inverse association with thyroid dysfunction risk. Similarly, CD4 count was not a significant predictor ($\beta = 0.0001$, $p = 0.913$), showing no meaningful contribution despite its univariate correlation in prior analysis.

Among the thyroid-related predictors, TSH ($\beta = +0.035$, $p = 0.588$), free T3 ($\beta = -0.219$, $p = 0.333$), and free T4 ($\beta = +0.543$, $p = 0.347$) also failed to reach statistical significance, as all 95% confidence intervals crossed zero, as shown in Table 6. This indicates that when modeled together, none of these variables independently explained the variability in thyroid dysfunction risk post-ART.

Table 3: Thyroid hormone levels before and after 3 months of ART

Parameter	*Baseline (mean ± SD)*	*Post-ART (mean ± SD)*	*t-value*	*p-value*
Free T3 (pg/mL)	2.92 ± 0.88	2.45 ± 1.00	2.877	0.005
Free T4 (ng/mL)	1.19 ± 0.37	1.10 ± 0.38	0.965	0.337
TSH (µIU/mL)	4.23 ± 4.13	7.50 ± 7.85	–6.518	**<0.001**

Bold value shows the marked significance of the value

Table 4: Distribution of patients according to thyroid status before and after ART

Thyroid status	*Baseline (n = 100)*	*Post-ART (n = 100)*	*p-value*
Euthyroid	62 (62.0%)	41 (41.0%)	*X* = 9.10 *p* = 0.028
Subclinical hypothyroidism	11 (11.0%)	17 (17.0%)	
Clinical hypothyroidism	14 (14.0%)	19 (19.0%)	
Subclinical hyperthyroidism	13 (13.0%)	23 (23.0%)	

Table 5: Correlation of CD4 count with thyroid parameters before and after ART

Correlation pair	*Spearman's ρ*	*p-value*	*95% CI (lower–upper)*	*Interpretation*
Baseline CD4 vs baseline TSH	–0.28	0.004	–0.45 to –0.09	Significant negative correlation
Baseline CD4 vs Baseline free T3	+0.35	0.001	+0.16 to +0.52	Significant positive correlation
Baseline CD4 vs Baseline free T4	+0.31	0.002	+0.12 to +0.49	Moderate positive correlation
Post-ART CD4 vs post-ART TSH	–0.34	0.001	–0.51 to –0.15	Stronger negative correlation post-ART
Post-ART CD4 vs post-ART free T3	+0.41	<0.001	+0.23 to +0.57	Strong positive correlation
Post-ART CD4 vs post-ART free T4	+0.36	0.001	+0.18 to +0.53	Significant positive correlation
ΔCD4 vs ΔTSH	–0.30	0.003	–0.48 to –0.10	CD4 rise linked to TSH decline
ΔCD4 vs Δfree T3	+0.33	0.002	+0.14 to +0.51	Immune recovery linked to T3 increase
ΔCD4 vs Δfree T4	+0.29	0.005	+0.10 to +0.47	Mild but significant association

Table 6: Multivariate logistic regression for predicting thyroid dysfunction post-ART (n = 100)

Predictor	*Coefficient (β)*	*Standard error*	*z-value*	*p-value*	*95% CI (lower–upper)*
Intercept	–0.180	1.263	–0.142	0.887	–2.66 to 2.30
Age (years)	–0.010	0.018	–0.562	0.574	–0.046 to 0.026
CD4 count	0.0001	0.0009	+0.109	0.913	–0.0017 to 0.0019
TSH (µIU/mL)	+0.035	0.064	+0.541	0.588	–0.091 to 0.160
Free T3 (pg/mL)	–0.219	0.227	–0.968	0.333	–0.663 to 0.225
Free T4 (ng/mL)	0.543	0.578	+0.940	0.347	–0.589 to 1.675

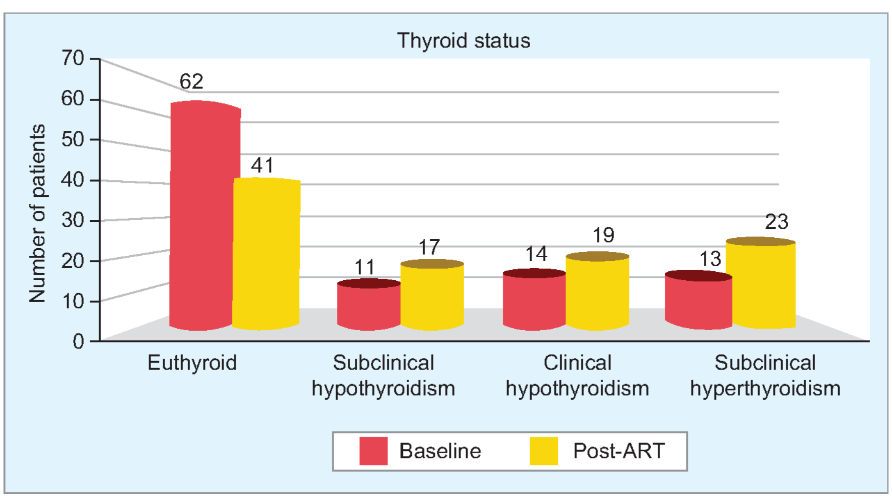
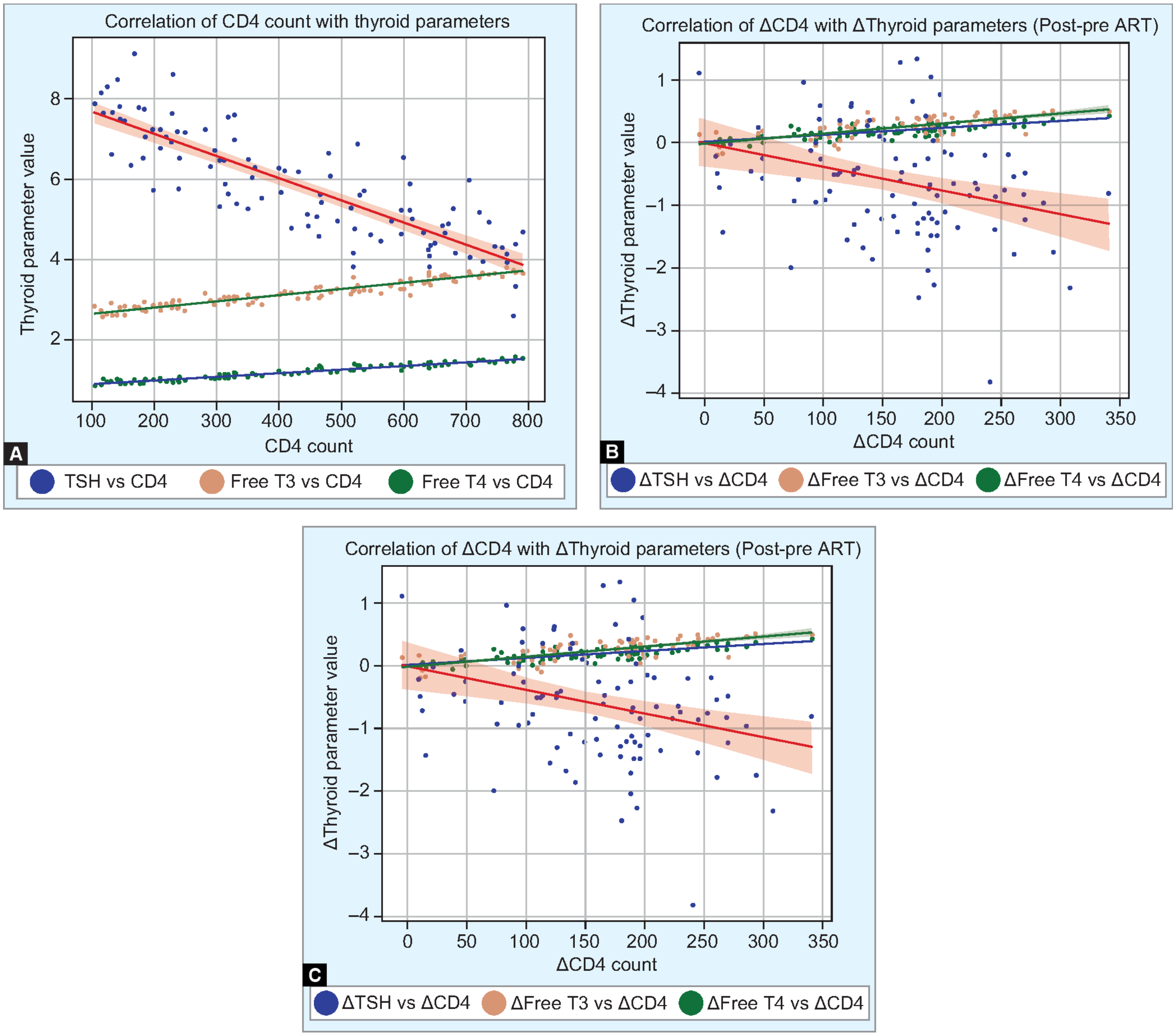


Fig. 4: Graphical representations of distribution of patients according to thyroid status before and after ART

Discussion

The purpose of this research is to measure thyroid function in newly diagnosed HIV patients before starting ART and to analyze changes in thyroid hormone levels following 3 months of treatment. We aimed to investigate how ART affects thyroid function in the early phases of treatment by examining important markers like TSH, free T3, and free T4. Our results contribute to the ongoing debate on the necessity of routine thyroid monitoring in people with HIV, especially when initiating ART.

The bulk of participants in our cohort were young to middle-aged individuals, with the largest percentage (34%) in the 20–29 age range and 25% in the 30–39 age range. Dev et al.[4] closely match the mean age of our



Figs 5A to C: (A) Graphical representations of correlation of CD4 count with thyroid parameters before and after ART; (B) Graphical representations of post-ART correlation of CD4 count with thyroid parameters (with random variation); (C) Graphical representations of correlation of ΔCD4 with thyroid parameters (post–pre ART)

sample, indicating that HIV infection primarily affects people in their early to midadult years.

After 3 months of ART, our study observed that TSH levels significantly increased (4.23 ± 4.13 to 7.50 ± 7.85 μIU/ml, $p < 0.001$) and free T3 levels significantly decreased (2.92 ± 0.88 to 2.45 ± 1.00 pg/mL, $p = 0.005$), indicating possible hypothyroid or subclinical thyroid dysfunction. Free T4 levels remained largely constant. These changes highlight the importance of regular thyroid monitoring in HIV patients receiving ART.

Similarly, Nasution et al.[5] documented comparable alterations in thyroid function in their cohort, where TSH levels rose from 1.44 ± 0.68 μIU/mL to 1.76 ± 0.91 μIU/mL following 3 months of ART, while mean free T4 levels dropped from 1.03 ± 0.14 ng/dL to 0.87 ± 0.13 ng/dL ($p = 0.006$). These changes were statistically significant, lending further support to the potential thyroid function alterations observed in our study. Like our results, their findings point to a trend toward elevated TSH and decreased free T4 during ART, which may indicate subclinical thyroid disease or early-stage hypothyroidism.

Dev et al.[4] emphasized the significance of monitoring TSH levels in HIV-positive patients, particularly when subclinical hypothyroidism is suspected. According to Bongiovanni et al.,[6] Surks et al.,[7] and Kong et al.,[8] TSH levels above 10 mU/L should be treated, while values between 4.5 and 10 mU/L may also require intervention.

According to Dutta and Kalita,[9] thyroid dysfunction was present in 36.84% of their HIV-infected patients, with subclinical hypothyroidism being the most prevalent anomaly. Overt hypothyroidism was more common in women (42.3%) than in men (35.8%). These results align with the higher TSH and lower free T3 levels observed in our study after ART, particularly among patients with thyroid dysfunction.

The impact of ART on thyroid function was further highlighted by our study, which revealed a significant shift in thyroid status after therapy. Subclinical hyperthyroidism increased from 13.0 to 23.0%, subclinical hypothyroidism from 11.0 to 17.0%, and clinical hypothyroidism from 14.0 to 19.0%, while euthyroid patients decreased from 62.0 to 41.0%. These findings are consistent with Madeddu et al.,[10] who reported a similar pattern of elevated subclinical hypothyroidism in ART-treated HIV patients.

Our observation of a rise in hypothyroid cases after ART aligns with Dev et al.,[4] who reported a high frequency of thyroid abnormalities (75.5%) in HIV-positive patients, with subclinical hypothyroidism being the most prevalent (53%). Although the percentage of overt hypothyroidism was higher in our cohort, Dev et al.[4] reported a lower incidence of overt hypothyroidism (8.4%), consistent with our observed increase in clinical hypothyroidism. Additionally, Dutta and Kalita[9] found a greater prevalence of subclinical hypothyroidism in HIV-positive individuals, supporting our findings of thyroid dysfunction after ART.

Conclusion

This prospective observational study aimed to assess thyroid function in newly diagnosed HIV patients and to evaluate the effect of ART over a period of 3 months. Specifically, the study focused on measuring serum free T3, free T4, and TSH levels at baseline and after ART initiation, and analyzing their relationship with immunological status as indicated by CD4 count. The study population consisted of 100 individuals with a mean age of 37.29 ± 13.01 years, predominantly male (70%). At baseline, 62% of patients were euthyroid, while the remaining 38% showed thyroid dysfunction, primarily subclinical hypothyroidism and subclinical hyperthyroidism. Following 3 months of ART, the prevalence of thyroid dysfunction increased: euthyroid patients decreased to 41%, and cases of subclinical hypothyroidism, clinical hypothyroidism, and subclinical hyperthyroidism rose noticeably. This shift in thyroid status distribution was statistically significant (Chi-squared test, p = 0.028), suggesting a potential impact of ART on thyroid physiology.

In terms of hormone levels, the study observed a statistically significant increase in mean TSH values (from 4.23 ± 4.13 µIU/mL to 7.50 ± 7.85 µIU/mL, $p < 0.001$) and a significant decrease in free T3 levels (from 2.92 ± 0.88 pg/mL to 2.45 ± 1.00 pg/mL, $p = 0.005$) post-ART. Free T4 levels did not show a significant change (p = 0.337). These results align with existing literature suggesting that ART may unmask or exacerbate subclinical thyroid dysfunction, possibly through immune reconstitution or direct effects on thyroid metabolism.

Correlation analysis demonstrated a significant negative association between CD4 count and TSH levels both before and after ART (ρ = −0.28 and −0.34, respectively) and a positive correlation between CD4 and both free T3 and free T4. These findings indicate that patients with lower immune function may be more prone to thyroid abnormalities and that immune recovery post-ART may influence thyroid hormone trends.

In conclusion, this study establishes that ART is associated with significant changes in thyroid hormone profiles, particularly an increase in TSH and a decline in free T3 levels, reflecting emerging thyroid dysfunction. The results underscore the importance of regular thyroid function monitoring in HIV patients, particularly after initiating ART, to facilitate early detection and management of evolving endocrine disturbances.

Limitations

The study was conducted with a relatively small sample size of 100 participants, which limits the generalizability of the findings to a larger population.

Thyroid function was only assessed after 3 months of ART, which may not be sufficient to observe long-term effects or trends in thyroid dysfunction.

Other potential confounding variables, such as nutrition, lifestyle factors, and comorbidities, were not systematically controlled for, which may have influenced thyroid function independently of ART.

Although the study evaluated changes pre- and post-ART, it did not provide a longitudinal analysis of thyroid function over an extended period, limiting insights into the long-term effects of ART on thyroid health.

Recommendations

Future studies should include a larger sample size to improve statistical power and ensure that the findings are more broadly applicable to diverse populations.

Longer follow-up periods should be implemented to better understand the sustained effects of ART on thyroid function and to detect any long-term thyroid dysfunction.

The inclusion of additional confounders, such as dietary habits, comorbid conditions, and concurrent medications, should be considered to better isolate the effects of ART on thyroid health.

A more comprehensive, multicenter approach could enhance the generalizability of the findings, allowing for a broader range of HIV patients from different geographical and socioeconomic backgrounds.

Public and Patient Involvement

During the study, all subjects were educated about thyroid dysfunction and the effects of ART on thyroid function. The results of the study were shared with HIV support groups to create awareness about thyroid dysfunction in HIV patients, and the importance of proper screening of thyroid function was explained to all HIV patients on ART.

References

1. Meissner ME, Talledge N, Mansky LM. Molecular biology and diversification of human retroviruses. Front Virol 2022;2:872599.
2. Baumgartner C, Da Costa BR, Collet TH, et al. Thyroid function within the normal range, subclinical hypothyroidism, and the risk of atrial fibrillation. Circulation 2017;136(22):2100–2116.
3. Bartalena L, Gallo D, Tanda ML. Autoimmune thyroid diseases. In: Rose NR, Mackay IR, editors. The Rose and Mackay Textbook of Autoimmune Diseases. 1st ed. Academic Press; 2024. pp. 561–584.
4. Dev N, Sahoo R, Kulshreshtha B, et al. Prevalence of thyroid dysfunction and its correlation with CD4 count in newly-diagnosed HIV positive adults–a cross-sectional study. Int J STD AIDS 2015;26(13):965–970.
5. Nasution MS, Lindarto D, Kembaren T. Effect of antiretroviral therapy to thyroid function status on new stage1 and 2 human immunodeficiency virus patient. Open Open Access Maced J Med Sci 2023;11(B):376–379.
6. Bongiovanni M, Adorni F, Casana M, et al. Subclinical hypothyroidism in HIV-infected subjects. J Antimicrob Chemother 2006;58(5):1086–1089.
7. Surks MI, Ortiz E, Daniels GH, et al. Subclinical thyroid disease: scientific review and guidelines for diagnosis and management. JAMA 2004;291:228–238.
8. Kong WM, Sheikh MH, Lumb PJ, et al. A 6-month randomized trial of thyroxine treatment in women with mild subclinical hypothyroidism. Am J Med 2002;112:348–354.
9. Dutta SK, Kalita BC. Thyroid function in newly diagnosed HIV-positive patients. J Assoc Physicians India 2023;71(5):11–12.
10. Madeddu G, Spanu A, Chessa F, et al. Thyroid function in human immunodeficiency virus patients treated with highly active antiretroviral therapy (HAART): a longitudinal study. Clin Endocrinol 2006;64(4):375–383.