Research Article

Journal of Clinical Virology and Translational Research

Thyroid Hormone Profile in HIV, Hepatitis B Virus, and Hepatitis C Virus Mono- and Co-Infected Patients in Edo State, Nigeria

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Received: August 05, 2026; **Accepted:** August 18, 2026; **Published:** August 25, 2026

Abstract

Background: Thyroid dysfunction is increasingly recognized as a complication of chronic viral infections, particularly HIV, hepatitis B virus (HBV), and hepatitis C virus (HCV). The interaction between viral infections and the hypothalamic-pituitary-thyroid axis remains incompletely understood, especially in sub-Saharan African populations. This study evaluated thyroid hormone profiles in mono- and co-infected patients compared with healthy controls in Edo State, Nigeria.

Methods: A cross-sectional study involving 660 subjects was conducted across four hospitals in Edo State. Study groups comprised 100 HIV-positive subjects on HAART, 50 HIV HAART-naïve subjects, 100 HBV-positive subjects, 100 HCV-positive subjects, 50 HBV/HCV co-infected subjects, 50 HIV/HBV co-infected subjects, 50 HIV/HCV co-infected subjects, and 100 apparently healthy controls. Plasma thyroid stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) were determined by enzyme immunoassay (EIA). Data were analyzed using one-way ANOVA and Duncan's Multiple Range post-hoc test at $p \leq 0.05$.

Results: TSH levels were significantly elevated in HIV subjects on HAART (4.16±0.7 mIU/L) and HBV (5.23±0.7 mIU/L) and HCV (4.08±0.8 mIU/L) subjects compared with controls (3.10±0.7 mIU/L; $p<0.05$), but significantly decreased in HIV HAART-naïve (2.47±1.0), HBV/HCV co-infected (2.98±0.8), HIV/HBV co-infected (2.09±0.7), and HIV/HCV co-infected (2.12±1.1) subjects. T3 levels were significantly decreased in HIV on HAART (0.56±0.2 ng/dl), HBV (0.54±0.1 ng/dl), and HCV (0.43±0.1 ng/dl) subjects compared with controls (1.53±0.3 ng/dl), but significantly elevated in all co-infected groups. T4 levels were significantly decreased in HIV and HBV mono-infected subjects but markedly elevated in all co-infected groups (HBV/HCV: 12.93±2.2; HIV/HBV: 12.46±3.2; HIV/HCV: 12.88±2.0 µg/dl) compared with controls (6.81±1.9 µg/dl; $p<0.05$).

Conclusion: Viral infections significantly alter thyroid hormone profiles, with distinct patterns observed between mono-infected and co-infected subjects. HAART-treated HIV subjects show elevated TSH suggesting subclinical hypothyroidism, while co-infected subjects demonstrate a paradoxical hyperthyroid-like pattern with elevated T3 and T4. These findings highlight the need for routine thyroid function monitoring in viral infection patients, particularly those with co-infections.

Keywords: Thyroid hormones, TSH, T3, T4, HIV, Hepatitis B, Hepatitis C, Co-infection, HAART, Nigeria

Introduction

Thyroid dysfunction is one of the most prevalent endocrinopathies in HIV-infected individuals. According to several studies, undiagnosed thyroid dysfunction, including subclinical hypothyroidism, is linked to severe morbidity and reduced quality of life [1-4]. It is estimated that up to 35% of all HIV-positive individuals experience subtle thyroid impairment [5-7]. Conversely, overt thyroid dysfunction is thought to affect 1% to 2% of individuals overall [6,7].

According to various studies conducted worldwide, the

Citation: Iweka FK, Omolumen LE, Okogun GRA, Iyevhobu KO, Dada F, et al. Thyroid Hormone Profile in HIV, Hepatitis B Virus, and Hepatitis C Virus Mono- and Co-Infected Patients in Edo State, Nigeria. J Clin Virol Trans Res. 2026. 1(1): 1-6. DOI: doi.org/10.61440/JCTV.2026.v1.02

prevalence of overt primary hypothyroidism in the general population and among HIV-positive individuals is 0.3% and 0–2.6%, respectively [6-9]. Similarly, other investigations have found that the prevalence of subclinical hypothyroidism is higher in HIV-positive individuals than in the general population (4.3% versus 3.5–12.2%) [6-9]. Unlike HIV, where the majority of cases are thought to have non-autoimmune origins, the majority of hypothyroidism in the general population is thought to have an autoimmune etiology [7,9].

Thyroid dysfunction is far more common (approximately 36–37%) in HIV-positive patients than in the general population, according to numerous studies [10,11]. However, according to several researchers, the morbidity of overt thyroid dysfunction in HIV-positive patients is comparable to that of the general population [7,10,12-14]. Thus, more investigation is needed to determine the frequency of thyroid dysfunction in HIV-positive individuals.

The natural progression of HCV infection is altered by HIV infection in several ways. HIV-positive patients have higher amounts of HCV RNA [15]. Patients with co-infections of HIV and HCV experience faster progression of liver disease than those with HCV infection alone [16,17]. HIV and HCV monoinfections both increase oxidative stress, which subsequently exacerbates liver fibrosis [18].

Hepatitis C virus infection has been associated with thyroid disease. According to earlier research, HCV infection may raise the risk of thyroid disease and even thyroid carcinoma [19-22]. The relationship between HCV and thyroid dysfunction remains controversial, with mixed findings across studies.

The hypothalamic-pituitary-thyroid axis may be impacted by the comorbidities that patients with HIV frequently have, such as infections and cancers linked to high levels of stress. This can complicate the interpretation of thyroid function tests. Research on the function of micronutrients in infectious diseases has produced a body of evidence that can be used to create therapeutic and preventive interventions.

Despite the recognized importance of thyroid function in viral infections, there is a paucity of data from Nigeria, particularly regarding the comparative assessment of thyroid hormone profiles across mono- and co-infected populations. This study was designed to determine the concentrations of thyroid hormones (TSH, T3, and T4) in HIV, HBV, and HCV mono- and co-infected subjects compared with healthy controls in Edo State, Nigeria.

Materials and Methods

Study Design and Population

This cross-sectional study was conducted in Edo State, Nigeria, involving 660 subjects recruited from Irrua Specialist Teaching Hospital, Central Hospital Uromi, Central Hospital Auchi, and Stella Obasanjo Hospital, Benin City. The study groups comprised 100 HIV-positive subjects on HAART, 50 HIV HAART-naïve subjects, 100 HBV-positive subjects, 100 HCV-positive subjects, 50 HBV/HCV co-infected subjects, 50 HIV/HBV co-infected subjects, 50 HIV/HCV co-infected subjects, and 100 apparently healthy controls aged 20–60 years.

Ethical Considerations

Ethical approval was obtained from the Edo State Ministry of Health Ethics Review Committee and the Ambrose Alli University Ethics Review Committee. Written informed consent was obtained from all participants.

Selection Criteria

Inclusion criteria included confirmed HIV, HBV, or HCV positive status and provision of informed consent. Exclusion criteria comprised sickle cell anemia, diabetes mellitus, hypertension, smoking, alcohol consumption, pregnancy, and refusal to consent.

Sample Collection

Blood samples were collected into lithium heparin containers and centrifuged at 3,000 rpm for 5 minutes. Plasma was separated and stored frozen prior to analysis.

Determination of Thyroid Hormones

Total T3 Concentration

Plasma total T3 was quantitatively determined using enzyme immunoassay (EIA) [23]. The assay principle involves competitive binding between patient serum T3 and T3-horseradish peroxidase conjugate for limited anti-T3 antibody binding sites on microtiter wells coated with goat anti-mouse IgG. After incubation and washing, TMB reagent was added, and absorbance was measured at 450 nm. T3 concentration was determined from a standard curve.

Total T4 Concentration

Plasma thyroxine (T4) was determined using enzyme immunoassay (EIA) [23]. The assay utilizes labeled T4 and T4 antibody with 8-anilino-1-naphthalene sulfonic acid (ANS) to prevent T4 binding to serum proteins. A predetermined quantity of labeled T4 competes with unlabeled T4 in the sample for binding sites on the T4 antibody. After separation of bound and unbound fractions, the labeled bound fraction was measured.

Thyroid Stimulating Hormone (TSH)

Plasma TSH was quantitatively determined by ELISA [24]. The assay uses a sandwich format with mouse monoclonal anti-TSH antibody immobilized on microtiter wells and horseradish peroxidase-conjugated goat anti-TSH antibody. TSH molecules are sandwiched between the solid phase and enzyme-linked antibodies. After incubation and washing, TMB reagent was added, and absorbance was measured at 450 nm.

Statistical Analysis

Data were analyzed using SPSS version 20.0. One-way ANOVA was used for group comparisons, with Duncan's Multiple Range post-hoc test applied where ANOVA showed significance at $p<0.05$. Results are presented as mean ± standard deviation.

Results

Thyroid Hormone Profiles in Mono- and Co-Infected Subjects

Table 1 presents the plasma concentrations of TSH, T3, and T4 across all study groups.

Thyroid Stimulating Hormone (TSH): TSH levels showed a bimodal distribution across study groups. Significant elevations were observed in HIV subjects on HAART (4.16±0.7 mIU/L; p<0.001), HBV subjects (5.23±0.7 mIU/L; p<0.001), and HCV subjects (4.08±0.8 mIU/L; p<0.001) compared with controls (3.10±0.7 mIU/L). Conversely, significant decreases were observed in HIV HAART-naïve (2.47±1.0 mIU/L; p<0.001), HBV/HCV co-infected (2.98±0.8 mIU/L; p=0.396, non-significant), HIV/HBV co-infected (2.09±0.7 mIU/L; p<0.001), and HIV/HCV co-infected (2.12±1.1 mIU/L; p<0.001) subjects.

Triiodothyronine (T3): T3 levels were significantly decreased in HIV subjects on HAART (0.56±0.2 ng/dl), HBV subjects (0.54±0.1 ng/dl), and HCV subjects (0.43±0.1 ng/dl) compared with controls (1.53±0.3 ng/dl; p<0.001). In contrast, all co-infected groups showed significant elevations: HIV HAART-naïve (2.72±1.1 ng/dl), HBV/HCV (2.52±0.2 ng/dl), HIV/HBV (2.54±0.2 ng/dl), and HIV/HCV (1.69±0.5 ng/dl) subjects.

Thyroxine (T4): T4 levels were significantly decreased in HBV subjects (5.77±1.0 µg/dl; p<0.001) compared with controls (6.81±1.9 µg/dl). HIV subjects on HAART (6.34±1.8 µg/dl), HIV HAART-naïve (6.16±0.9 µg/dl), and HCV subjects (6.35±1.5 µg/dl) showed marginal decreases that did not reach statistical significance in all pairwise comparisons. Strikingly, all co-infected groups demonstrated marked T4 elevation: HBV/HCV (12.93±2.2 µg/dl), HIV/HBV (12.46±3.2 µg/dl), and HIV/HCV (12.88±2.0 µg/dl), all significantly above control values (p<0.001).

Table 1: Thyroid Function Profile in HIV, HBV, HCV Mono- and Co-Infected Subjects

Parameters	Control (n=100)	HIV/ HAART (n=100)	HIV Naïve (n=50)	HBV (n=100)	HCV (n=100)	HBV/ HCV (n=50)	HIV/HBV (n=50)	HIV/HCV (n=50)	f-value	p-value
TSH(mIU/L)	3.10±0.7^{a}	4.16±0.7^{b}	2.47±1.0^{c}	5.23±0.7^{d}	4.08±0.8^{b}	2.98±0.8^{a}	2.09±0.7^{c}	2.12±1.1^{c}	146.59	0.000*
T3 (ng/dl)	1.53±0.3^{a}	0.56±0.2^{b}	2.72±1.1^{c}	0.54±0.1^{b}	0.43±0.1^{b}	2.52±0.2^{c}	2.54±0.2^{c}	1.69±0.5^{a}	14.91	0.000*
T4 (µg/dl)	6.81±1.9^{a}	6.34±1.8^{ab}	6.16±0.9^{bcd}	5.77±1.0^{c}	6.35±1.5^{ad}	12.93±2.2^{e}	12.46±3.2^{e}	12.88±2.0^{e}	194.39	0.000*

Values with different superscripts are significantly different at p≤0.05.

Post-Hoc Pairwise Comparisons

Table 2 presents the detailed post-hoc analysis for thyroid hormone comparisons.

Table 2: Post-Hoc Analysis of Thyroid Function Profile

Comparison	TSH	T3	T4	Inference
Control vs HIV on HAART	0.000	0.001	0.082	Significant
Control vs HIV Naïve	0.000	0.001	0.046	Significant
Control vs HBV	0.000	0.001	0.000	Highly Significant
Control vs HCV	0.000	0.000	0.081	Significant
Control vs HBV/HCV	0.396	0.006	0.000	Significant
Control vs HIV/HBV	0.000	0.004	0.000	Highly Significant
Control vs HIV/HCV	0.000	0.653	0.000	Significant
HIV/HAART vs HIV Naïve	0.000	0.000	0.565	Significant
HIV/HAART vs HBV	0.000	0.945	0.031	Significant
HIV/HAART vs HCV	0.478	0.659	0.907	Significant
HIV/HAART vs HBV/HCV	0.000	0.000	0.000	Highly Significant
HIV/HAART vs HIV/HBV	0.000	0.000	0.000	Highly Significant
HIV/HAART vs HIV/HCV	0.000	0.002	0.000	Highly Significant
HIV Naïve vs HBV	0.000	0.000	0.235	Significant
HIV Naïve vs HCV	0.000	0.000	0.552	Significant
HIV Naïve vs HBV/HCV	0.001	0.501	0.000	Significant
HIV Naïve vs HIV/HBV	0.018	0.635	0.000	Significant
HIV Naïve vs HIV/HCV	0.028	0.014	0.000	Significant
HBV vs HCV	0.000	0.710	0.031	Significant
HBV vs HBV/HCV	0.000	0.000	0.000	Highly Significant
HBV vs HIV/HBV	0.000	0.000	0.000	Highly Significant
HBV vs HIV/HCV	0.000	0.001	0.000	Highly Significant

HCV vs HBV/HCV	0.000	0.000	0.000	Highly Significant
HCV vs HIV/HBV	0.000	0.000	0.000	Highly Significant
HCV vs HIV/HCV	0.000	0.000	0.000	Highly Significant
HBV/HCV vs HIV/HBV	0.000	0.893	0.210	Significant
HBV/HCV vs HIV/HCV	0.000	0.043	0.994	Significant
HIV/HBV vs HIV/HCV	0.870	0.046	0.211	Significant

Discussion

This study provides comprehensive evidence of significant thyroid hormone alterations in HIV, HBV, and HCV mono- and co-infected subjects in Edo State, Nigeria. The findings reveal distinct patterns of thyroid dysfunction that have important clinical implications.

The elevated TSH levels observed in HIV subjects on HAART are consistent with previous studies. Beltran et al. (2006) and Thongam et al. (2015) also found higher levels of TSH among HAART users [25,26]. Following the initiation and use of HAART, autoimmune reconstitution inflammatory syndrome may be linked to elevated blood TSH levels in those receiving treatment [10,27]. Stavudine, a component of some HAART regimens, has been directly linked to thyroid hormone synthesis and metabolism disturbances [5,6,11,25].

Conversely, the low serum TSH levels in the HAART-naïve group may have been caused by high viral loads prior to starting HAART, which resulted in TSH level depletion [26,28,29]. The elevated TSH in HBV and HCV mono-infected subjects suggests that chronic hepatitis independently affects the hypothalamic-pituitary-thyroid axis, possibly through inflammatory cytokine-mediated suppression of thyroid hormone synthesis or altered hepatic conversion of T4 to T3.

The markedly elevated TSH in HBV subjects (5.23±0.7 mIU/L) is consistent with Antonelli's (2004) suggestion that hypothalamic-pituitary dysfunction contributes to abnormal TSH secretion in advanced liver disease [20]. Since dopamine has been demonstrated to have an inhibitory influence on the control of TSH secretion, a lowered dopaminergic tone as a result of the buildup of false neurotransmitters may be the cause of elevated basal TSH concentrations [30].

The decreased T3 levels in HIV subjects on HAART, HBV, and HCV mono-infected subjects are consistent with the "low T3 syndrome" commonly observed in chronic illnesses. This pattern reflects impaired peripheral conversion of T4 to T3, often associated with decreased hepatic deiodinase activity in liver disease. The most prevalent alteration in plasma levels of thyroid hormones in hepatic disorders is a decrease in total T3 and FT3 concentrations, reported to be associated with the severity of hepatic dysfunction [31].

The paradoxical elevation of T3 and T4 in co-infected subjects represents a novel and clinically significant finding. All co-infected groups (HBV/HCV, HIV/HBV, HIV/HCV) demonstrated significantly elevated T3 and T4 compared with both controls and mono-infected subjects. This pattern suggests a hyperthyroid-like state in co-infected individuals, which may result from several mechanisms:

First, chronic immune activation in co-infection may stimulate thyroid hormone release through cytokine-mediated mechanisms. Second, altered hepatic metabolism in co-infected subjects may reduce thyroid hormone clearance, leading to accumulation. Third, the severe oxidative stress documented in co-infected subjects may directly affect thyroid follicular cell function.

The marked T4 elevation in co-infected subjects (approximately 90% above control values) is particularly striking. Hepatocytes' basal metabolic rate is regulated by T4 and T3, which in turn affects hepatic function. The liver metabolizes thyroid hormones and controls their systemic endocrine effects [32]. In co-infected subjects with severely compromised liver function, impaired hormone metabolism may lead to accumulation.

The finding that 51% of HIV-infected subjects on HAART were diagnosed with thyroid dysfunctions (subclinical hypothyroidism and hyperthyroidism) is significantly higher than rates reported by Shujing et al. (2016) and Beltran et al. (2003), who reported 31.1% and 16%, respectively [6,33]. This discrepancy could be explained by the prolonged HIV infection duration in our sample. Compared to the HAART-naïve group, which had 3% and 9% of subclinical hypothyroidism and hyperthyroidism, respectively, the HAART group exhibited a higher prevalence of thyroid dysfunction.

The most prevalent thyroid malfunction among HIV-positive individuals was clinical hypothyroidism, which is in line with the findings of Beltran et al. (2003) [6]. However, Madeddu et al. (2003) found that among HIV patients using HAART, subclinical hypothyroidism was the most common thyroid impairment [7]. Hoffmann & Brown (2007) and Weetman (2014) observed that while the likelihood of aberrant thyroid hormone test findings was greater in HIV-positive HAART participants, the risk of clinical hypothyroidism in these individuals was comparable to that of the general population [10,14].

The relationship between thyroid hormones and HBV/HCV infections remains incompletely understood. According to some studies, HBV and HCV may affect the synthesis, movement, or clearance of thyroid hormones, which may have an effect on thyroid hormone metabolism. Although this view is not widely accepted, some propose that the immunological response to the virus may also play a role in the alteration of thyroid hormone levels [18].

The clinical implications of these findings are substantial. Elevated TSH in HAART-treated subjects suggests subclinical hypothyroidism, which may contribute to fatigue, weight gain, and cognitive impairment commonly reported by these patients. The hyperthyroid-like pattern in co-infected subjects may explain symptoms of anxiety, weight loss, and cardiac

arrhythmias observed in this population. Routine thyroid function monitoring should be incorporated into the standard care of all viral infection patients, with particular attention to co-infected individuals.

Conclusion

This study demonstrates that HIV, HBV, and HCV infections significantly alter thyroid hormone profiles, with distinct patterns between mono-infected and co-infected subjects. HAART-treated HIV subjects show elevated TSH suggesting subclinical hypothyroidism, while co-infected subjects demonstrate a paradoxical hyperthyroid-like pattern with markedly elevated T3 and T4. These findings highlight the need for routine thyroid function monitoring in viral infection patients, particularly those with co-infections, to enable early detection and management of thyroid dysfunction.

Routine screening of thyroid function (TSH, T3, and T4) should be incorporated into the standard care of all HIV, HBV, and HCV infected patients. Particular attention should be paid to co-infected patients, who demonstrate the most marked thyroid hormone alterations. HAART-treated HIV patients with elevated TSH should be evaluated for subclinical hypothyroidism and considered for thyroxine supplementation if clinically indicated. Co-infected patients with elevated T3 and T4 require monitoring for hyperthyroidism-related complications including cardiac arrhythmias and bone loss.

Conflict of Interest

The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

Ethical Permission

Ethical approval was obtained from the University Ethics Committee and also informed consent was sought from the subjects before collection of blood samples.

Funding

This research did not receive any grant from funding agencies in the public, commercial, or not-for-profit sectors.

Acknowledgements

The authors would like to acknowledge the management of Ambrose Alli University (AAU), Ekpoma, Edo State, Nigeria for creating the enabling environment for this study. Thanks to all the Laboratory and technical staff of Saint Kenny Diagnostic and Research Centre, Ekpoma, Edo State, Nigeria for their excellent assistance and for providing medical writing support/ editorial support in accordance with Good Publication Practice (GPP3) guidelines.

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