Archives of Current Research International

Volume 24, Issue 5, Page 758-768, 2024; Article no.ACRI.118592
ISSN: 2454-7077

Assessment of Thyroid Profiles (TSH, T3 and T4) in HIV Positive Patients at Central Hospital, Benin City, Nigeria

Omolumen, L.E. [a], Iweka, F.K. [a], Iyevhobu, K.O. [b,c*], Airhomwanbor, K.O. [a], Usiobeigbe, O.S. [a], Adelakun, A.A. [d], Bisiriyu, A.H. [e], Ikede, R.E. [f], Ugege, C. [g], Oboh, M.M. [h], Asibor E. [i], Adeji, A.J. [j], Obohwemu, K.O. [k], Oikerhe, E.G. [h], and Akhigbe, A.M. [a]

[a] Department of Chemical Pathology, Faculty of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[b] Department of Medical Microbiology, Faculty of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[c] St Kenny Research Consult, Ekpoma, Edo State, Nigeria.
[d] Department of Medical Laboratory Science, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria.
[e] Nigeria Field Epidemiology and Laboratory Training Program (NFELTP), Nigeria.
[f] Department of Bacteriology, Federal School of Medical Laboratory Technology, Jos, Plateau State, Nigeria.
[g] Department of Medical Laboratory Science, Edo University, Uzairue, Edo State, Nigeria.
[h] Department of Haematology and Blood Transfusion Science, Faculty of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[i] Department of Histopathology and Cytopathology, Faculty of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[j] Department of Microbiology, School of Applied Science and Technology, Auchi Polytechnic Auchi, Edo State, Nigeria.
[k] Department of Health, Wellbeing and Social Care, Global Banking School, Birmingham, United Kingdom.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: https://doi.org/10.9734/acri/2024/v24i5751

Open Peer Review History:

**Corresponding author: Email: kennylamai@yahoo.com;*

Cite as: *L.E., Omolumen, Iweka, F.K., Iyevhobu, K.O., Airhomwanbor, K.O., Usiobeigbe, O.S., Adelakun, A.A., Bisiriyu, A.H., Ikede, R.E., Ugege, C., Oboh, M.M., Asibor E., Adeji, A.J., Obohwemu, K.O., Oikerhe, E.G., and Akhigbe, A.M. 2024. "Assessment of Thyroid Profiles (TSH, T3 and T4) in HIV Positive Patients at Central Hospital, Benin City, Nigeria". Archives of Current Research International 24 (5):758-68. https://doi.org/10.9734/acri/2024/v24i5751.*

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Original Research Article

Received: 18/04/2024
Accepted: 20/06/2024
Published: 27/06/2024

ABSTRACT

This study evaluated the thyroid profile (TSH, T3 and T4) of HIV positive subjects visiting Central Hospital in Benin City, Edo State. The subjects in this study comprise of HIV positive volunteers aged between 18 to 50 years attending Central Hospital Benin City, Edo State, Nigeria. A total of 120 subjects were recruited for this study. The study comprises of eighty HIV positive subjects and forty apparently healthy subjects (controls). HIV serostatus was determined according to centre for disease and prevention. Plasma total TSH, T4 & T3 were quantitatively determined using enzyme immunoassay. TSH levels were significantly higher ($p<0.05$) in subjects (2.75±1.59 miu/ml) when compared with the control (1.92±1.11 miu/ml). T3 levels were not significantly lower ($p>0.05$) in subjects (1.32±0.63 ng/ml) when compared with the control (1.49±0.35 ng/ml). T4 levels were significantly lower ($p<0.05$) in subjects (6.06±1.83 µg/dl) when compared with the control (7.09±1.78 µg/dl). The results showed a significant non-increased in TSH ($p>0.05$) in subjects who had been on drugs within 11-15years, when compared with others. T3 and T4 levels were higher ($p>0.05$) in subjects who had been on drugs within 16-20 years when compared with other subjects. In conclusion, TSH levels were significantly higher ($p<0.05$) in subjects when compared with the control, and T_4 levels were significantly lower ($p<0.05$) in subjects when compared with the control. Larger studies are needed to examine the epidemiology and health consequences of mild thyroid dysfunction in HIV-infected subjects and to better inform screening and treatment guidelines.

Keywords: Thyroid profile; TSH; T3; T4; HIV.

1. INTRODUCTION

Human Immunodeficiency Virus (HIV) infection, caused by a lentivirus within the Retroviridae family, leads to progressive impairment of the cellular immune system, increasing susceptibility to infections and tumors, and culminating in Acquired Immune Deficiency Syndrome (AIDS) [1]. AIDS was first identified as an emerging disease in the early 1980s and spread quickly over the world, changing over the course of two decades from a mysterious condition to a pandemic that affected tens of millions of people [2]. Only 24.5 million of the 37.9 million HIV/AIDS positive individuals worldwide, mostly in low- and middle-income nations, have access to antiretroviral medication (ART) [3]. These figures include 2.1 million children under the age of 15 who also live with the virus. With the third-largest HIV epidemic, India has 2.1 million HIV-positive people in 2015. The virus had spread from high-risk populations to the general public. HIV-positive people knew their status by 2017; among them, 56% were on antiretroviral therapy [3]. HIV-related endocrine disorders—thyroid dysfunction in particular—are becoming more widely recognized; cross-sectional investigations have shown that thyroid abnormalities are quite prevalent among HIV patients. The decrease in CD4+ count was linked to a progressive elevation in serum thyroxin-binding globulin (sr.TBG) as reported by Lambert et al. [4], without any equivalent rise in other binding proteins. Increased sr.TSH and s.TBG concentrations were linked to immunosuppression and CD4+ cell depletion, according to Feldt-Rasmussen et al. [5] who also reported low FT-4. Early in HIV infection, subtle changes in thyroid function test (TFT) results are common. Various mechanisms have been hypothesized to explain these changes, including direct HIV infection, tumors, cytokine effects, medication side effects, and direct thyroid infection by opportunistic pathogens [6].

Three deiodinase enzymes (Type 1 (D1), Type 2 (D2), and Type 3 (D3)) control thyroid hormone

metabolism. They are in charge of rT3 and T3 to T2 conversion, T4 to T3 activation, and T4 to rT3 inactivation. While D3, which is present in the liver, skin, and central nervous system (CNS), predominantly converts T4 to rT3 and T3 to T2, D1 is mostly found in the liver and kidney and contributes 30–40% of the generation of extra-thyroidal T3. Because of decreased D1 activity, cirrhosis patients have low total and free T3, elevated rT3, increased rT3 to T3 ratio, and increased T4 to rT3 conversion via D3 [7]. Low T3 levels lower hepatocyte basal metabolic rate and maintain liver function. Thyroid hormones control hepatic function and bilirubin metabolism [8]. Experimental animals undergoing thyrotoxicosis exhibit elevated bile bilirubin production, potentially due to heightened hepatic heme decomposition [9]. Unconjugated hyperbilirubinemia may be the result of thyroid hormone-induced changes in bilirubin metabolism, particularly decreased glucuronyltransferase activity, which may conceal Gilbert's syndrome. Additionally, bile acid synthesis and pool size are reduced by thyroid hormones [10]. Thyroid hormone metabolism and systemic effects are affected by liver illnesses; thyroid profile abnormalities have been reported in liver patients. Because of an increase in thyroid-binding globulin, an acute-phase reactant, acute hepatitis raises total T4 with normal free T4. The T3:rT3 ratio is adversely correlated with the severity of cirrhosis. Total and free T3 are lowered in cirrhosis, indicating a decrease in D1 activity and the conversion of T4 to T3 [10].

HIV infection continues to be a major public health concern in sub-Saharan Africa, particularly Nigeria, despite the fact that HAART has considerably decreased the disease's morbidity and death. HAART, however, raises the risk of diabetes and cardiovascular disease by being associated with metabolic abnormalities such lipodystrophy and dyslipidemia [11–16]. Although HIV patients frequently have abnormal thyroid function tests, overt thyroid problems do not differ considerably from the general population. As the disease advances, some asymptomatic HIV patients show changed thyroid hormone levels, but most have normal thyroid function [17–20].

HIV patients may have subclinical thyroid dysfunction, ill euthyroid syndrome, hyperthyroidism, or hypothyroidism. The immune system is impacted by these anomalies, which also drastically lower patients' quality of life [21–24]. Reduced peripheral T4 to T3 conversion, decreased production of thyrotropin-releasing hormone (TRH), and changes in thyroid-binding proteins are the mechanisms leading thyroid dysfunction in HIV patients [25–28]. Some stable HIV patients show asymptomatic abnormalities in thyroid function tests, such as low serum T4 levels; others show characteristic sick euthyroid syndrome patterns, such as low T3 and FT3 levels connected with deteriorating HIV disease [29–30]. Thyroid hormone production and peripheral conversion are impacted by these alterations, which are brought on by a decrease in TRH secretion and a reduction in T4 to T3 conversion [35–38]. This study aims to evaluate thyroid profiles in HIV-positive individuals in Benin City, Edo State, contributing to understanding thyroid dysfunction in HIV/AIDS.

2. MATERIALS AND METHODS

The study was conducted in Edo State's Benin-City. Benin City, the capital of Edo State in southern Nigeria, with an estimated population of 1,147,188 as of 2006. The city is situated about 25 miles to the north of the Benin River. It is located 200 km east of Lagos on the route. Nigeria's rubber business is centered on Benin, but another significant traditional sector is the extraction of oil from palm nuts. Benin City is located 80 meters above sea level and at 6.34 degrees North latitude and 5.63 degrees East longitude [39].

Study Population: The research subjects comprised HIV-positive volunteers aged between 18 to 50 years, drawn from Central Hospital Ekpoma, Edo State. A total of one hundred participants were recruited, including forty apparently healthy individuals as controls and eighty HIV-positive individuals as test samples. The sample size determination was based on a prevalence rate of 3.4% for HIV in Edo State, as reported by Onovo et al. [40]. Considering a 50% attrition rate, the initial sample size was upgraded to 80 subjects.

Research Design: The study adopted a prospective and cross-sectional design, aiming to assess the thyroid profile (TSH, T3, and T4) of HIV-positive subjects in Benin City, Edo State, Nigeria. Medical history, including age, gender, and other pertinent medical information, was collected from each participant's medical records. The study spanned a duration of six months [39-42]. To ensure the validity of HIV status, both confirmed positive and negative

cases were re-tested using HIV test strips and standard laboratory procedures. Based on this validation, HIV-positive and control subjects were selected and grouped for the study. Thyroid profile analysis, including TSH, T3, and T4, was conducted using venous blood samples collected from the ante-cubital vein.

Sample Collection: Five millilitres (5mls) of venous blood sample were collected from each subject from the ante-cubital vein using sterile disposable syringe. Blood samples were processed to separate serum from red cells and stored at -20°C pending analysis.

Sample Analysis: HIV serostatus was determined according to the CDC – UMD HIV rapid testing serial algorithm II guideline, utilizing the Determine HIV – ½ kit for initial testing. Positive results were confirmed using the Uni-Gold HIVI/II test kit, with discordant results resolved using a tie-breaker kit (Stat-Pak).

Furthermore, plasma total T3 and T4 levels were quantitatively determined using enzyme immunoassay techniques, while plasma TSH levels were quantified using ELISA. The immunoassay techniques involved competitive binding of labeled and unlabeled hormones to specific antibodies, with subsequent measurement of the labeled fraction to calculate hormone concentrations.

Guidelines for detection of thyroid dysfunction: Thyroid function categories were defined based on established criteria for hypothyroidism and hyperthyroidism, including primary, secondary, and subclinical forms of both conditions.

Data Analysis: Statistical analysis of the obtained results was performed using ANOVA (LSD) with significance set at $p < 0.05$ [42,43].

3. RESULTS

3.1 Socio-Demographic Characteristics of the Study Population

Table 1 revealed the socio-demographic characteristics of the study population. The subjects were categorized into five age groups; 16-25 years; 26-35 years; 36-45 years; 46-55 years and 56-65 years. The result for age showed that majority of the subjects were within the age range of 36-45 years accounting for 33.8%, followed by 46-55 years 23.8%, 26-35 years 18.8%, 16-25 years 15.0% and 56-65 years being the least accounted for 8.8%. The Age (Mean ±SD) of the subjects was (39.34±11.76). With respect to gender, 27.5% of the subjects were male and 72.5% of the subjects were female. Based on duration of drugs, 8.8% of the subjects have been on drugs within the period of 0-11months, 1-5years 33.8%, 6-10years 32.5%, 11-15years 18.8% and 16-20years 6.3%.

3.2 TSH, T_3 and T_4 Levels between Subjects and Control

The results in Table 2 showed the comparison of TSH, T_3 and T_4 levels between the subjects and control. The result showed that TSH levels were significantly higher ($p<0.05$) in subjects (2.75±1.59 miu/L) when compared with the control (1.92±1.11 miu/L). On the contrary, T_3 levels were also not significantly lower ($p>0.05$) in subjects (1.32±0.63 ng/L) when compared with the control (1.49±0.35 ng/L). T_4 levels were significantly lower ($p<0.05$) in subjects (6.06±1.83 ng/L) when compared with the control (7.09±1.78 ng/L).

3.3 TSH, T_3 and T_4 Levels between Male Subjects and Male Control

The results in Table 3 showed the comparison of TSH, T_3 and T_4 levels between the male subjects and control. The result showed that TSH levels were not significantly higher ($p>0.05$) in male subjects (2.59±1.58 miu/L) when compared with the control (2.32±1.23 miu/ L). T_3 levels were not significantly lower ($p>0.05$) in male subjects (1.33±0.65 ng/L) when compared with the control (1.54±0.38 ng/L). T_4 levels were also not significantly lower ($p>0.05$) in male subjects (6.27±1.88 ng/L) when compared with the control (6.58±1.70 ng/L).

3.4 TSH, T_3 and T_4 Levels between Female Subjects and Female Control

The results in Table 4 showed the comparison of TSH, T_3 and T_4 levels between the female subjects and control. The result showed that TSH levels were significantly higher ($p<0.05$) in female subjects (2.81±1.60 miu/L) when compared with the control (1.47±0.76 miu/ L). On the contrary, T_3 levels were not significantly lower ($p>0.05$) in female subjects (1.32±0.64 ng/L) when compared with the control (1.44±0.32 ng/ L). T_4 levels were significantly lower ($p<0.05$)

in female subjects (5.98±1.83 ng/L) when compared with the control (7.65±1.75 ng/L).

3.5 TSH, T_3 and T_4 levels of the Subjects with Respect to Gender

The results in Table 5 showed the comparison of TSH, T_3 and T_4 levels of the subjects with respect to gender. The result showed that TSH levels were not significantly lower (p>0.05) in male subjects (2.59±1.58 miu/ L) when compared with the female subjects (2.81±1.60 miu/ L). T_3 levels were not significantly higher (p>0.05) in male subjects (1.33±0.65 ng/ L) when compared with the female subjects (1.32±0.64 ng/ L). T_4 levels were also not significantly higher (p>0.05) in male subjects (6.27±1.88 ng/ L) when compared with the female subjects (5.98±1.83 ng/ L).

3.6 TSH, T_3 and T_4 levels of the Subjects According to Age

The results in Table 6 showed the comparison of TSH, T_3 and T_4 levels of the subjects according to age. The results showed a significant non-increased in TSH (p>0.05) in subjects aged 16-25 years (3.04±1.26 miu/L) when compared with subjects within the age range of 46-55 years (3.01±1.92 miu/L), 36-45 years (2.95±1.69 miu/L), 56-65 years (2.30±0.49 miu/L) and 26-35 years (2.03±1.39 miu/L). T_3 levels were higher (p>0.05) within the age range of 26-35 years (1.69±0.73 ng/L) when compared with 56-65 years (1.31±0.68 ng/L), 16-25 years (1.25±0.55 ng/L), 46-55 years (1.24±0.67 ng/L) and 36-45 years (1.21±0.53 ng/L). T_4 levels were higher within the age range of 26-35 years (7.10±1.80 ng/L) when compared with 56-65 years (6.13±2.01 ng/L), 45-55 years (6.04±1.88 ng/L), 36-45 years (5.83±1.83 ng/L) and 16-25 years (5.22±1.24 ng/L).

3.7 TSH, T_3 and T_4 levels of the Subjects According to Duration of Drugs

The results in Table 7 showed the comparison of TSH, T_3 and T_4 levels of the subjects according to duration of drugs. The results showed a significant non increased in TSH (p>0.05) in subjects who had been on drugs within 11-15years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 16-20 years. T_3 levels were higher (p>0.05) in subjects who had been on drugs within 16-20 years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 11-15 years. T_4 levels were higher (p>0.05) in subjects who had been on drugs within 16-20 years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 11-15 years.

Table 1. Socio-demographic characteristics of the study population (n=120)

Variables		Frequency	Percentage (%)
Age (Years)	16-25	12	15.0
	26-35	15	18.8
	36-45	27	33.8
	46-55	19	23.8
	56-65	7	8.8
	Age (Mean ±SD)	39.34±11.76	
Gender	Male	22	27.5
	Female	58	72.5
Duration of drugs	0-11months	7	8.8
	1-5years	27	33.8
	6-10years	26	32.5
	11-15years	15	18.8
	16-20years	5	6.3

Table 2. TSH, T_3 and T_4 levels between subjects and control

Parameters	Control (n=40)	Subjects (n=80)	t value	P value
TSH (miu/L)	1.92±1.11	2.75±1.59	2.967	0.004
T_3 (ng/L)	1.49±0.35	1.32±0.63	1.560	0.121
T_4 (ng/L)	7.09±1.78	6.06±1.83	2.913	0.004

KEY: n=Sample size; p>0.05= Not significant; p<0.05= Significant

Table 3. TSH, T_3 and T_4 levels between male subjects and male control

Parameters	Male Control (n=21)	Male Subjects (n=22)	t value	P value
TSH (miu/L)	2.32±1.23	2.59±1.58	0.619	0.539
T_3 (ng/L)	1.54±0.38	1.33±0.65	1.279	0.208
T_4 (ng/L)	6.58±1.70	6.27±1.88	0.571	0.571

KEY: n=Sample size; p>0.05= Not significant; p<0.05= Significant

Table 4. TSH, T_3 and T_4 between female subjects and female control

Parameters	Female Control (n=19)	Female Subjects (n=58)	t value	P value
TSH (miu/L)	1.47±0.76	2.81±1.60	3.502	0.001
T_3 (ng/L)	1.44±0.32	1.32±0.64	0.771	0.443
T_4 (ng/L)	7.65±1.75	5.98±1.83	3.481	0.001

KEY: n=Sample size; p>0.05= Not significant; p<0.05= Significant

Table 5. TSH, T_3 and T_4 levels of subjects with respect to gender

Parameters	Male Subjects (n=22)	Female Subjects (n=58)	t value	P value
TSH (miu/L)	2.59±1.58	2.81±1.60	0.557	0.579
T_3 (ng/L)	1.33±0.65	1.32±0.64	0.063	0.950
T_4 (ng/L)	6.27±1.88	5.98±1.83	0.615	0.540

KEY: n=Sample size; p>0.05= Not significant; p<0.05= Significant

Table 6. TSH, T3 and T4 levels of the subjects according to age

Parameters	16-25 Years (n=21)	26-35 Years (n=29)	36-45 Years (n=26)	46-55 Years (n=23)	56-65 Years (n=11)	F-value	P-value
TSH (miu/L)	3.04±1.26 ^{a}	2.03±1.39 ^{a}	2.95±1.69 ^{a}	3.01±1.92 ^{a}	2.30±0.49 ^{a}	1.255	0.295
T_3 (ng/L)	1.25±0.55 ^{a}	1.69±0.73 ^{a}	1.21±0.53 ^{ab}	1.24±0.67 ^{ab}	1.31±0.68 ^{a}	1.645	0.172
T_4 (ng/L)	5.22±1.24 ^{a}	7.10±1.80 ^{b}	5.83±1.83 ^{a}	6.04±1.88 ^{ab}	6.13±2.01 ^{ab}	2.226	0.074

Table 7. TSH, T3 and T4 levels of the subjects according to duration of drugs

Parameters	0-11 Months (n=21)	1-5 Years (n=29)	6-10 Years (n=26)	11-15 Years (n=23)	16-20 Years (n=11)	F value	P value
TSH (miu/L)	2.75±1.25 ^{a}	2.83±1.73 ^{a}	2.56±1.37 ^{a}	3.33±1.86 ^{a}	1.53±0.78 ^{ab}	1.370	0.253
T_3 (ng/L)	1.36±0.63 ^{a}	1.20±0.71 ^{a}	1.39±0.58 ^{a}	1.27±0.65 ^{a}	1.74±0.31 ^{a}	0.884	0.478
T_4 (ng/L)	5.34±2.61 ^{a}	5.83±1.74 ^{a}	6.26±1.65 ^{a}	6.28±1.73 ^{a}	6.60±2.63 ^{a}	0.609	0.658

KEY: n=Sample size; p>0.05= Not significant; p<0.05= Significant

Values in a row with the same superscript are not significantly different at p<0.05

4. DISCUSSION

The study aims at evaluating the thyroid profile (TSH, T3 and T4) of HIV positive subjects visiting Central Hospital in Benin City, Edo State. In our study majority of the subjects were within the age range of 36-45 years accounting for 33.8%, followed by 46-55 years 23.8%, 26-35 years 18.8%, 16-25 years 15.0% and 56-65 years being the least accounted for 8.8%. The Age (Mean ±SD) of the subjects was (39.34±11.76). This is in agreement with the study of Shukla et al [44] in which majority of the cases (81.45%) were between 20-45 years and Michèle et al. [45] in which mean age was 40.8 years (SD = 9.54). The mean age of participants was similar to that observed in previous studies [23, 46,47].

With respect to gender, 27.5% of the subjects were male and 72.5% of the subjects were female. Based on duration of drugs, 8.8% of the subjects have been on drugs within the period of 0-11months, 1-5years 33.8%, 6-10years 32.5%, 11-15years 18.8% and 16-20years 6.3%. The

result from this study showed that TSH levels were significantly higher ($p<0.05$) in subjects (2.75±1.59 miu/L) when compared with the control (1.92±1.11 miu/L). On the contrary, T_3 levels were also not significantly lower ($p>0.05$) in subjects (1.32±0.63 ng/L) when compared with the control (1.49±0.35 ng/L). T_4 levels were significantly lower ($p<0.05$) in subjects (6.06±1.83 ng/L) when compared with the control (7.09±1.78 ng/L). According to Palanisamy et al. [48], thyroid dysfunction is common in HIV infection, and as the disease progresses, people with HIV infection experience a primary hypothyroid-like stage [49]. As a result, serum TSH, free T3, and free T4 can all be utilized as surrogate indicators of the disease's advancement. Thyroid dysfunction may be a sign of HIV severity or progression, according to Thongam et al. [32]. 14% of Thai children with HIV in a Panamonta et al. research [50] had low serum T3, normal TSH, and FT4 values consistent with sick-euthyroidism, and all of them were clinically euthyroid.

Though the exact etiology of thyroid malfunction is unknown, possible explanations include medication responses, autoimmune illness, concurrent infections, and opportunistic infections that cause damage [51]. The advancement of the disease is linked to thyroid abnormalities, which include high viral loads and significant immunosuppression [52,53]. As the condition progresses, serum levels of TBG gradually rise [54,55]. Although the cause of the rise in TBG is unclear, it does not appear to be connected to either protein clearance or serum estrogen levels [56].

On the duration of HAART, the results showed a significant non-increased in TSH ($p>0.05$) in subjects who had been on drugs within 11-15years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 16-20 years. T_3 levels were higher ($p>0.05$) in subjects who had been on drugs within 16-20 years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 11-15 years. T_4 levels were higher ($p>0.05$) in subjects who had been on drugs within 16-20 years, when compared with subjects who have been on drugs for a period of 0-5 months, 1-5 years, 6-10 years and 11-15 years. Rather than the Immune Reconstitution syndrome, a prior study linked the raised levels of thyroid autoantibodies to some general effects of enhanced B-cell activation observed during the clinical course of HIV infection [57].

Thyroid abnormalities with HIV infection include pathological alterations as well as disruptions in thyroid function [6,53]. Our current research demonstrates that thyroid dysfunction is common in HIV infection and that patients with progressive HIV infection experience a subclinical hypothyroid-like stage as their illness progresses [58]. As the condition progresses, a number of thyroid function tests, including FT3/FT4 and serum TSH, can be utilized as a surrogate measure. When treating an HIV infection, one should wait to begin replacement medication because the condition may respond to very vigorous antiretroviral therapy. The actual picture of thyroid abnormalities in HIV/AIDS patients is provided by our current study. In conclusion, patients with HIV infection and AIDS frequently experience aberrant TFTs. There are management guidelines (see above) for overt dysfunction. Larger research is yet required to assess the frequency and consequences of moderate thyroid dysfunction in HIV-positive individuals and to develop screening and treatment protocols.

When diagnosing thyroid diseases in individuals exhibiting thyroid-related symptoms or nonspecific systemic symptoms, thyroid function testing is a suitable diagnostic procedure. Thyroid function testing for asymptomatic people is controversial, nevertheless, and affects both the general public and HIV-positive patients. The high frequency of subclincal hypothyroidism and the possible benefit of levothyroxine medication in this population may warrant monitoring older patients regardless of HIV status [59]. The pathophysiology of subclincal hypothyroidism may differ in HIV-infected patients, despite cross-sectional studies reporting a higher prevalence than typically seen in the general population. Currently, there is not enough evidence to support routine screening of all HIV-infected individuals. Comparably, even though it occurs frequently, an isolated low FT4 level has no known repercussions and shouldn't be the focus of routine monitoring [60].

The classic sick euthyroid state may be a manifestation of concomitant morbidities or stress connected to advanced disease. This is likely caused by a hypothalamic-pituitary deficit linked to the progression of immunodeficiency and cachexia [41]. Thyroid function may be impacted by stavudine used to treat HIV infection, but the current study is unable to prove this correlation due to the small number of individuals using the medication.

5. CONCLUSION

Test results for thyroid function are frequently abnormal in HIV-positive people. This is particularly true while on HAART, when the prevalence of subclinical hypothyroidism and immunological reconstitution may cause Graves' disease, and when isolated low FT4 values seem to be more common. As of right now, there is not enough data to support the screening of asymptomatic HIV-positive people for thyroid abnormalities. The results of this study demonstrate that, in comparison to the control, participants' TSH levels were significantly higher ($p<0.05$) and their T4 levels were significantly lower ($p<0.05$). To further inform screening and treatment recommendations, it is advised that additional research be conducted with a reasonable sample size to examine the epidemiology and health effects of moderate thyroid dysfunction in HIV-positive individuals.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

CONSENT

Only already diagnosed and confirmed HIV positive subjects and apparently healthy subjects who had no known medical condition were recruited for this study. This study specifically included only subjects between 18-50 years who gave consent for the study.

Individuals who are not within the age range of 18-50 years whose HIV status has not been confirmed were excluded. Control subjects who show any sign of visible ailments, would not give consent and have any underlying illness such as diabetes, cardiovascular, sickle cell, pregnant and renal diseases were also excluded from this study.

ETHICAL APPROVAL

Ethical permission for this study was obtained from the Edo State Hospital Management Board, Benin City, Edo State, Nigeria. Informed consent was obtained from the patients prior to the collection of samples for this study. The purpose of the study was exhaustively explained to the patients and assured of the confidentiality of the information obtained from them.

ACKNOWLEDGEMENTS

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

COMPETING INTERESTS DISCLAIMER

Authors have declared that they have no known competing financial interests or non-financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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