Prevalence of thyroid dysfunction among HIV-positive pregnant women attending antenatal care at Kakamega county general referral hospital, Kenya

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ABSTRACT

Thyroid dysfunction during pregnancy increases risks for both mothers and infants, with Human Immunodeficiency Virus (HIV) infection further complicating thyroid physiology through immune and metabolic changes. Despite the high HIV burden in sub-Saharan Africa, few studies address thyroid dysfunction among HIV-positive pregnant women. This study assessed the prevalence of thyroid dysfunction among HIV-positive pregnant women in Kakamega County, Kenya. A facility-based cross-sectional study was conducted at Kakamega County General Referral Hospital (KCGRH). Sixty-seven women (HIV-positive and HIV-negative pregnant women and non-pregnant controls) were enrolled. Serum thyroid stimulating hormone (TSH), free triiodothyronine (fT3), and free thyroxine (fT4) were measured using ELISA. Data analysis employed descriptive statistics, Mann–Whitney U, Kruskal–Wallis, and Spearman correlation tests in SPSS v26. Ethical approval and informed consent were obtained from HIV-positive pregnant women, among whom 50.0% exhibited thyroid dysfunction, all presenting with hyperthyroidism. HIV-negative pregnant women had a higher prevalence (72.7%), comprising hyperthyroidism (63.6%), hypothyroidism (9.1%), and euthyroidism (13.6%). HIV-positive women had significantly elevated systolic blood pressure (p=0.044). Thyroid dysfunction, predominantly hyperthyroidism, is common among HIV-positive pregnant women in Kakamega. The integration of thyroid screening into ANC and HIV programs is recommended.

Keywords: HIV, Hyperthyroidism, Kakamega CGRH, Pregnancy, Thyroid Dysfunction

I. INTRODUCTION

Thyroid gland is located in the anterior neck, it produces trioodothyronine (T3), thyroxine (T4) and calcitonin. The three play a vital role in the metabolic processes, growth, neurological development and the cardiovascular functions and serum calcium regulation by the calcitonin as per (Allen & Fingeret, 2025). Triiodothyronine and the Thyroxine are synthesized by the follicular cells, while the calcitonin is secreted by the parafollicular C-cells which helps in regulating the calcium levels by inhibiting bone resorption (Li *et al*., 2024). Thyroglobulin mimics the function of the precursor for thyroid hormone synthesis and its measurement is valuable in monitoring thyroid cancer recurrence. Thyroid disorders are among the most common endocrine diseases across the world and common among women of reproductive age (Gao *et al*., 2024, Nigatie *et al*., 2024).

Pregnancy induces physiological changes which affect the thyroid function with potential cause of disorders such as hyperthyroidism and hypothyroidism (Puthiyachirakal *et al*., 2025, Vamja *et al*., 2024, Nazarpour *et al*,. 2016). These disorders increase risks of miscarriage, preeclampsia, intrauterine growth restriction, stillbirth and neurodevelopmental impairment in infants (Galofre & Davies, 2009).

Global thyroid dysfunction has a prevalence of 7.5% (Hu *et al*., 2022) but highest in African countries especially those with low and income social demographic countries. It above 20% as per (Vargas-Uricoechea *et al*., 2025) with

higher prevalence of upto 7 folds among women compared to men, it affects 5-10% of adults as per (Garmendia Madariaga *et al.*, 2014). In Sub-Saharan Africa, the prevalence varies widely depending on varied factors with rates as high as 90% in iodine-deficient populations (Ogbera & Kuku, 2011). Thyroid dysfunction is as well highly reported among people living with HIV (PLWH), particularly those on antiretroviral therapy (ART), The mechanisms include immune reconstruction inflammatory syndrome, chronic inflammation and ART induced effects (Pillay *et al*., 2020).In Kenyan study reported thyroid dysfunction prevalence of 51.9% among HIV-infected patients, especially those on ART (Adan *et al*., 2025).

1.1 Statement of the Problem

Pregnancy significantly alters the physiological demand on the thyroid gland. This leads to higher production of the thyroid hormones to enable it meet the fetal and maternal needs (Puthiyachirakal *et al*., 2025, Alemu *et al*., 2016). The rise in thyroid function is physiologically linked to the increased thyroid-binding globulin levels and the stimulatory effect of human chorionic gonadotrophin (hCG) on thyroid-stimulating hormone receptors (Lee & Pearce, 2022).

Studies done reports show that the thyroid production may increase by as much as 50% during gestation (Singh *et al.*, 2025). The early pregnancy symptoms can resemble those of thyroid dysfunction, hence pausing a risk to misdiagnosis and inadequate treatment (Lee & Pearce, 2022). Failure to identify and manage the maternal thyroid disorders can result into adverse outcome; miscarriage, preterm birth, neurodevelopmental impairments, stunted growth in the fetus (Alves Junior et al., 2022), (Grob et al., 2025), (Vamja *et al*., 2024) Alemu *et al*., 2016).

Routine screening of thyroid function hormones during pregnancy is therefore essential to safeguard maternal and fetal health (Singh *et al.*, 2025).Thyroid function tests are the essential biomarkers for diagnosing and differentiating thyroid disorders. The key biomarkers include Thyroid Stimulating Hormone (TSH), Free thyroxine (FT4), Free triiodothyronine (FT3), thyroglobulin (Tg) and thyroid autoantibodies such as TPOab and TgAb (Soh & Aw, 2019) (Carvalho & Drobrzenski, 2023).TSH remains the most sensitive single marker for primary hypothyroidism and hyperthyroidism, whereas FT4 and FT3 measurements are valuable for assessing disease severity (Jimoh *et al.*, 2020). In pregnancy, thyroid assessment is particularly important because maternal thyroid hormones are vital for fetal neurodevelopment, especially in the first trimester when the fetus fully depends on the maternal supply (Moog *et al.*, 2015, Nazarpour *et al*., 2016). The physiological rise in hCG stimulates thyroid hormones production often causing lower TSH levels during early gestation (Lee & Pearce, 2022). Differentiating gestational transient thyrotoxicosis from Graves' disease is crucial to avoid unnecessary treatment (Menconi *et al.*, 2014).

Despite these findings, little is known about thyroid dysfunction among HIV-positive pregnant women in Kenya. Therefore, this study aimed to assess the prevalence of thyroid dysfunction among HIV-positive pregnant women who attend antenatal care at Kakamega County General Referral Hospital. Early recognition and treatment of thyroid abnormalities are essential to prevent complications such as metabolic syndrome, cardiovascular risk, and adverse pregnancy outcomes in HIV-positive women of reproductive age (Dutta & Kalita, 2023, Galofre & Davies, 2009).

1.2 Research Objective

To assess the prevalence of thyroid dysfunction among HIV-positive pregnant women attending antenatal care at Kakamega County General Referral Hospital.

II. LITERATURE REVIEW

2.1 General Background on Thyroid Hormones

The thyroid gland, located in the anterior neck, produces triiodothyronine (T3), thyroxine (T4), and calcitonin, which play essential roles in metabolism, growth, neurological development, and cardiovascular function (Allen & Fingeret, 2025). T3 and T4 are synthesized by follicular cells, whereas calcitonin is secreted by parafollicular C-cells and helps regulate calcium levels by inhibiting bone resorption (Shahid *et al*., 2025). Thyroglobulin acts as a precursor for thyroid hormone synthesis, and its measurement is valuable in monitoring thyroid cancer recurrence (Li *et al*., 2024).

The hypothalamic–pituitary–thyroid (HPT) axis regulates hormone production through thyroid-stimulating hormone (TSH), which controls T3 and T4 release (Blick *et al*., 2015). Optimal thyroid function is crucial for reproduction, cognitive function, and metabolic balance. Dysregulation can lead to hypothyroidism, hyperthyroidism, and thyroid nodules, all of which may affect fertility, cardiovascular health, and pregnancy outcomes (Kalra *et al.*, 2013).

2.2 Thyroid Function Biomarkers

Thyroid function tests (TFTs) are essential in diagnosing and differentiating thyroid disorders. Key biomarkers include TSH, free thyroxine (FT4), free triiodothyronine (FT3), thyroglobulin (Tg), and thyroid autoantibodies such as TPOAb and TgAb (Carvalho *et al.*, 2013) TSH remains the most sensitive single marker for primary hypothyroidism and hyperthyroidism, whereas FT4 and FT3 measurements are valuable for assessing disease severity (Jimoh *et al*., 2020).

In pregnancy, thyroid assessment is particularly important because maternal thyroid hormones are vital for fetal neurodevelopment, especially in the first trimester when the fetus depends entirely on maternal supply (Nazarpour *et al*., 2016). The physiological rise in human chorionic gonadotropin (hCG) stimulates thyroid hormone production, often causing lower TSH levels during early gestation (Lee & Pearce, 2022). Differentiating gestational transient thyrotoxicosis from Graves' disease is crucial to avoid unnecessary treatment (Menconi *et al.*, 2014).

2.3 Thyroid Dysfunction and Pregnancy

Pregnancy imposes significant physiological demands on the thyroid gland, increasing hormone requirements by up to 50% (Singh *et al*., 2025). Elevated estrogen levels lead to higher thyroid-binding globulin concentrations, and hCG directly stimulates TSH receptors, increasing T3 and T4 production (Alemu *et al*., 2016). These changes may unmask or worsen pre-existing thyroid conditions. Untreated hypothyroidism in pregnancy is associated with miscarriage, preterm delivery, preeclampsia, and impaired neurocognitive outcomes in offspring (Andersen *et al*., 2014). Hyperthyroidism, most commonly caused by Graves' disease, may result in gestational hypertension, low birth weight, and fetal thyrotoxicosis if poorly controlled (Galofre & Davies, 2009). Given the overlap between pregnancy symptoms and thyroid disorders, screening and timely intervention are crucial to prevent maternal and fetal complications (Nazarpour et al., 2016).

Thyroid dysfunction is increasingly reported among people living with HIV (PLWH), particularly those on antiretroviral therapy (ART). The mechanisms include immune reconstitution inflammatory syndrome, direct viral effects on the thyroid, and drug-induced thyroid toxicity (Chen *et al*., 2020). Hypothyroidism is the most frequent presentation, often subclinical, whereas hyperthyroidism can occur during immune reconstitution (Beltran *et al.*, 2003). A study in South Africa reported a significant prevalence of thyroid abnormalities in HIV-infected individuals with comorbid diabetes mellitus, emphasizing the need for targeted screening (Pillay *et al*., 2020). Similarly, Kenyan data from Meru County highlight a substantial burden of thyroid dysfunction among PLWH, suggesting that routine thyroid function tests could improve early detection and clinical management (Adan *et al*., 2025). Early recognition and treatment of thyroid abnormalities are essential to prevent complications such as metabolic syndrome, cardiovascular risk, and adverse pregnancy outcomes in HIV-positive women of reproductive age (Galofre & Davies, 2009).

III. METHODOLOGY

3.1 Study Design, Site and Population

This was a comparative cross-sectional study conducted at KCGRH ANC department that targeted pregnant women attending antenatal clinic, while control population were patient care takers who met inclusion criteria. Kakamega CGRH is located in Kakamega town of Western part of Kenya. KCGRH reports an annual ANC pregnant attendance of 2,976 mothers which ensured an adequate sample size for statistical power. Kakamega CGRH is a level 5 Referral facility hence serves as a major maternal health facility for the county and surrounding regions which captures diverse cases from both rural and urban setup, enriched with various consultants to attend to every case. The diagnostic infrastructure was well established with basic laboratory ISO 15189:2012 and 2022 certified, available imaging capacity good enough to conduct maternal thyroid function studies and any radiological diagnostics. The facility supports researches with existing protocols, ethics review committee and competent personnel to support in clinical studies.

3.2 Sample size and Sampling Technique

The study population consisted of pregnant mothers who were both HIV positive and negative attending Ante Natal Clinic at Kakamega CGRH of ages 18-45. The Kakamega CGRH ANC attendance had set targets at the year 2016/2017 of 248 per month and 2976 per year, however the study aimed at the calculated population only, which was a representation of the annual target population. Sample size of Sixty Seven (67) women (34 pregnant, 33 controls) participants were consecutively recruited through sample size calculation using the formula for comparing two independent proportions.

$n = Z^2[p_1(1-p_1) + p_2(1-p_2)]$

$n = Z^2[p_1(1-p_1) + p_2(1-p_2)] / d^2$

$n = \{(0.083(1-0.083) + 0.381(1-0.381))(1.96)^2\} / (0.298)^2$

$n=14$

Where:

n was sample size

p_1 was the estimated thyroid dysfunction prevalence for study population. This study used 8.3%, the prevalence of transient gestational thyroid dysfunction among 16-week pregnant women attending KNH reported by (Ndungu *et al.,* 2009).

p_2 The estimated prevalence for the non-pregnant group was 38.1% as calculated from the findings from a study by (Ngugi, 2014) at Kenyatta National Hospital.

Z was the standard normal variate for the chosen α (the significance level or critical P value). In this study α was 5% (P<0.05) and therefore Z = 1.96

d was the percentage allowable margin of error =-29.8%=-0.298

Since the number of eligible participants was too small, the finite population correction (FPC) was applied

$n_{fpc} = n / (1 + (n - 1)/N)$

Where:

n is the calculated sample size

N is the study population calculated for 1 month

n_{fpc} is the estimated finite population correction

Substituting n=14 and N=248 gave an adjusted sample size of 13.3, which was rounded up to 14.

Attribution rate:

$n_{final} = n_{adj} / (1 - r)$

Where:

n_{adj} is the adjusted sample size from the formula and the FPC=28

r is the attrition rate 15%

Calculated n_{final} was 32.9 rounded up to 33

Estimated sample size was 67

3.3 Ethics consideration

Ethical approval and permit to conduct the study were obtained from Masinde Muliro University of Science and Technology Scientific and Ethical Review Committee (REF: MMU/COR: 403012vol2 (6)) and National Commission for Science, Technology and Innovation (license no: NACOSTI/P/24/33856) and Kakamega county ethical review board. Informed consent obtained from study participants. Confidentiality was ensured by using password protected computer system only accessible to the investigators. Qualified medical and research personnel were utilized in the study ensuring utmost integrity of participants' information and also following of the Nuremberg ethical conduct.

3.4 Blood Sample Collection

The blood specimens for analysis were collected by the venipuncture through the median cubital veins and median basilic veins depending on the client's visibility of the identified veins. Vein visibility was enhanced through torniquet procedure and clear swabbing using alcohol swabs for disinfections. Four milliliters of blood samples were drawn and collected in a red top vacutainer using closed system as a sample safety phlebotomy procedure and biosafety practice. The redtop vacutainer hastens clotting processes, then centrifuged at 1000rpm for 2 minutes to obtain clear serum, which was the appropriate state of blood for the assays.

3.5 Laboratory Analysis

Blood tests was performed on the acceptable blood serum for thyroid function are TSH, fT_4 and fT_3. (British thyroid foundation of 2015. ELISA procedure by the STAT FAX 303 Plus equipment which involves manual preparation and incubation followed by automated absorbance reading at 450nm wavelength was utilized to analyze thyroid function tests. Thyroid hormone concentrations were calculated using a standard curve generated by the STAT FAX 303 plus equipment. ELISA is the gold standard diagnostic for the detection and quantification of biomarkers. Venous blood samples were analyzed for TSH, fT3, and fT4 using ELISA kits. Reference ranges: TSH: 0.32–5.2 mIU/L; fT3: 1.9–4.3 pg/ml; fT4: 0.7–1.8 ng/dl.

3.6 Method Validation

The test procedure followed the Standard Operating Procedures for both the equipment and the tests. During phlebotomy process, the blood samples were collected 4 mls of blood using closed system to red top vacutainer which have clot activator gel that had not been opened or expired. The blood was allowed to clot then centrifuge at 1000rpm for 2 minutes to obtain clear serum. The serum was analyzed against their respective quality control samples that are pre-coated in the wells of the kits as prescribed by the manufacturer. Storage of the serum from the samples was at 2-8ºC for archival of samples that had been analyzed for 7days after collection and at -20ºC for those analyzed after 7 days of collection, to enable any further quality checks.

The STAT FAX 303 plus equipment was well serviced and maintained as per the manufacturer's requirements. The equipment operation requirements for accuracy were met which included operation within temperatures of 18^0C - 35^0C with operational temperatures monitors. Calibrators, controls and blank indicators were analyzed and met satisfactory requirements prior performing the tests and results printout reviewed and filed (Stat Fax 303 Plus Operator's Manual Pdf Download | ManualsLib). Reagents used for testing were from ELISA kit from Pishtazteb Diagnostics whose accuracy ranges from 90%-100% for TSH, fT4, fT3. The reagents were stored as per the manufacturers' recommendation and expiry dates were monitored.

3.7 Statistical Analysis

Data Collection was done using a ruled paper that was divided into name, sex, age, parity, gravidity, weight, systolic blood pressure, diastolic blood pressure, HIV test results, TSH, fT_3, fT_4 data from all the study population and control group were captured and documented in hardcopy. Data were then transferred onto an excel sheet in soft copy for analysis then coded, entered and cleaned in a Microsoft Excel and safely stored in a password-protected computer. These were then imported into and analyzed using Statistical Package for Social Sciences (SPSS) software version 26.0 (Inc Chicago, IL). Descriptive statistics was used to describe the general distribution of TSH, fT_3, fT_4. These included medians and interquartile ranges and presented as tables and figures. Wilcoxon rank sum test / Mann-Whitney u test was used to compare the study group and the control group, the HIV positive pregnant women and HIV naive pregnant women for a significant difference. Kruskal Wallis was used to compare 3 independent variables (the three trimester). *P*- Value < 0.05 was considered statistically significant. Spearman's correlations were used to identify factors that influenced thyroid function biomarkers and the demographics correlations. Thyroid function tests reference ranges were: TSH (0.32mU/L-5.2mU/L), free T3 (1.9-4.3Pg/ml), free T4 (0.7ng/dl-1.8ng/dl).

IV. FINDINGS & DISCUSSION

4.1 Circulating Thyroid Hormones Levels among HIV Positive Pregnant Women

As shown in *Table 1* below, The HIV-positive pregnant group (n < 12) exhibited distinct thyroid hormone patterns compared to both HIV-negative pregnant women and non-pregnant controls. The median TSH level among HIV-positive pregnant women was 2.55 IU/L (IQR: 0.90), which was not significantly different from the HIV-negative pregnant group (P = 0.957) or the non-pregnant control group (P = 0.154). This finding indicates that HIV infection, in this study population, did not significantly alter pituitary-thyroid axis regulation during pregnancy. For free triiodothyronine (fT3), HIV-positive pregnant women had a median level of 3.93 pmol/L (IQR: 1.19), which was slightly higher than that of HIV-negative pregnant women (3.77 pmol/L), although this difference was not statistically significant (P = 0.575). However, when compared to non-pregnant women, the increase in fT3 levels was highly significant (P < 0.0001). These results suggest an overall pregnancy-related enhancement of peripheral thyroid hormone production that is not significantly affected by HIV status but may be slightly accentuated in HIV-positive women.

Free thyroxine (fT4) levels were also slightly elevated among HIV-positive pregnant women (median: 9.33 pmol/L) compared to HIV-negative pregnant (9.01 pmol/L) and non-pregnant controls (9.01 pmol/L), though the differences did not reach statistical significance (P = 0.471 and P = 0.203, respectively). This indicates that HIV-positive status does not significantly impact circulating fT4 concentrations in pregnancy. Interestingly, the fT3/fT4 ratio, a marker of peripheral conversion of thyroxine to triiodothyronine, was higher among HIV-positive pregnant women (median: 0.46) compared to controls (0.33), and this difference was statistically significant (P = 0.011). This finding implies an enhanced peripheral conversion process, which may represent a compensatory mechanism to maintain adequate circulating levels of the biologically active T3 hormone during pregnancy despite potential metabolic stress related to HIV infection. Overall, the results demonstrate that HIV-positive pregnant women exhibit a pattern of normal TSH and fT4 levels, elevated fT3 levels compared to non-pregnant women, and a significantly increased fT3/fT4 ratio, suggesting increased conversion of T4 to T3. These findings are consistent with physiological adaptation to pregnancy but may indicate slightly more pronounced metabolic activation in HIV-positive women, possibly related to immune activation or ART-induced metabolic changes.

Table 1

Thyroid Hormone Profile in HIV-Positive Pregnant Women

All maternal units	***TSH, IU/L***	***fT_3, pmol/L***	***fT_4, pmol/L***	***fT_3/fT_4***
HIV-negative pregnant, n<22	2.50 (1.40)	3.77 (1.58)	9.01 (2.09)	0.44 (0.24)
1st trimester, n<4	2.00 (2.40)	3.00 (0.96)	9.59 (2.51)	0.34 (0.16)
2nd trimester, n<11	2.20 (1.00)	4.47 (2.16)	9.27 (1.16)	0.45 (0.19)
3rd trimester, n<7	2.90 (2.10)	3.70 (1.54)	8.62 (6.44)	0.52 (0.39)
[a]*p*-value	0.791	0.080	0.449	0.098
HIV-positive pregnant, n<12	2.55 (0.90)	3.93 (1.19)	9.33 (1.26)	0.46 (0.15)
[b]*p*-value	0.957	0.575	0.471	0.885
Non-pregnant, n<33	2.90 (1.70)	2.00 (0.40)	9.01 (1.80)	0.33 (0.10)
[c]*p*-value	0.090	**<0.0001**	0.600	**0.003**
[d]*p*-value	0.154	**<0.0001**	0.203	**0.011**

Data shown are medians with interquartile ranges in parentheses and numbers and percentages in parentheses for gravidity and parity. A comparison between trimesters of gestation. Comparison between HIV-negative pregnant and

HIV-positive pregnant women. Comparison between HIV-negative pregnant and non-pregnant women. Comparison between HIV-positive pregnant and non-pregnant women. Values in bold are significant P-values. Mann-Whitney The U test was used to compare two independent groups, and the Kruskal-Wallis test was used to compare trimesters. In reference to *table 1* above, when directly comparing HIV-positive to HIV-negative pregnant women, no significant differences were observed in TSH, fT3, or fT4 levels (P = 0.957, 0.575, and 0.471, respectively), indicating that HIV infection did not markedly alter overall thyroid function during pregnancy. On comparing the HIV-positive pregnant women to the control population, there was a significant difference for fT3 and fT3/fT4 ratio; TSH, fT3, fT4, and fT4/fT3 (P=0.154, P<0.001, P=0.203, P=0.011). However, the fT3/fT4 ratio was slightly higher in HIV-positive women (0.46 vs. 0.44), suggesting a trend toward enhanced peripheral conversion of T4 to T3 in this group. Although not statistically significant in this comparison, the finding aligns with the significantly elevated fT3/fT4 ratio observed between HIV-positive women and non-pregnant controls (P = 0.011). Clinically, this pattern may reflect an increased metabolic demand in HIV-positive pregnancies, possibly influenced by immune activation or the effects of antiretroviral therapy (ART) (Rubingh *et al*., 2020). These results underscore the importance of routine thyroid hormone monitoring in HIV-positive pregnant women, as subtle alterations in hormone dynamics could predispose to adverse maternal or fetal outcomes if left undetected (Adan *et al*., 2025 ; Pillay *et al*., 2020).

4.2 Association between Free Triiodothyronine (fT3) levels and the fT3/fT4 Ratio with Age, Weight and Blood Pressure among HIV-Positive Pregnant Women

The correlation was done using Spearman's rank correlation. Statistically, the index of peripheral conversion of fT4 to fT3 (FT3/FT4) and free triiodothyronine was significantly higher among HIV-positive pregnant women (P<0.001) as seen in *table 2.* There was very strong correlation between fT3 and fT3.fT4 ratio (ρ=0.880, P<0.001). There was positive correlation between age and diastolic blood pressure (ρ=0.615, P<0.033), systolic blood pressure and diastolic blood pressure (ρ=0.596, P<0.041).

Table 2

Association of Free T3 and Free T3/T4 Ratio with Age, Weight and Blood Pressure in HIV-Positive Pregnant Women

Analyte	Free T3		Free T3/T4		Age		Weight		Systolic Blood pressure		Diastolic Blood pressure	
	ρ	P	ρ	P	ρ	P	ρ	P	ρ	P	ρ	P
Free T3	1.000	-	0.880	**0.000**	0.071	0.827	-0.204	0.524	0.504	0.094	-0.440	0.153
Free T3/T4	0.880	**0.000**	1.000	-	-0.084	0.795	-0.217	0.499	-0.368	0.240	-0.486	0.109
Age	0.071	0.827	-0.084	0.795	1.000	-	0.123	0.704	0.367	0.240	0.615	**0.033**
Weight	-0.204	0.524	-0.217	0.499	0.123	0.704	1.000	-	0.627	0.029	0.392	0.207
Systolic Blood pressure	-0.504	0.094	0.368	0.240	0.367	0.240	0.392	0.207	1.000	-	0.596	**0.041**
Diastolic Blood pressure	-0.440	0.153	-0.486	0.109	0.615	**0.033**	0.627	0.029	0.596	**0.041**	1.000	-

Data presented are correlation coefficient (rho, p) with associated P values. Statistical analysis was performed using Spearman's rank correlation test. fT3: free Trioodothyronine.fT3/fT4: free triiodothyronine free thyroxine ration with age, blood pressure and weight. Correlation coefficient (rho, p) and associated P-values with significant correlation shown in bold. Significance was set at a value <0.05.

Elevated systolic blood pressure in HIV-positive women may be explained by combined vascular inflammation from HIV and hyperthyroidism-related cardiovascular strain (Irani *et al*., 2018). Regional differences in iodine sufficiency and socioeconomic status may explain the higher prevalence at KCGRH compared to Kenyatta National Hospital (Mungla, 2017). In general, hyperthyroidism was the main dysfunction in this study, and this compares with a retrospective study that was conducted in Accra Ghana (Ge *et al.*, 2024), whose hyperthyroidism was 67.0%, hypothyroidism (26.9%). This study contributes novel data on thyroid dysfunction in HIV-positive pregnancies in western Kenya. Limitations include small sample size and single-center scope. Larger, multicenter studies are warranted.

4.3 Pattern of Thyroid Dysfunction in Pregnancy with HIV

The graphical output was generated from descriptive analysis done by the IBM SPSS for the thyroid dysfunction patterns are shown in *Figure 1* below. The analysis of the thyroid dysfunction patterns was illustrated as percentages as follows; 73.5% (25 of 34) of the pregnant women: HIV-negative, 86.4% (19 of 22) and HIV-positive, 50.0% (6 of 12) presented with thyroid dysfunctions. Most of the thyroid dysfunctions among the HIV-negative pregnant women were

hyperthyroidism (63.6%) with 13.6% and 9.1% having euthyroidism and hypothyroidism, respectively. A half (50.0%) of the HIV-positive pregnant women had hyperthyroidism and none had euthyroidism or hypothyroidism. Blood pressure differences: HIV-positive women had higher systolic blood pressure (p=0.044). Correlations: In HIV-positive women, thyroid hormone correlations with age, weight, and blood pressure were weaker compared to HIV-negative women.

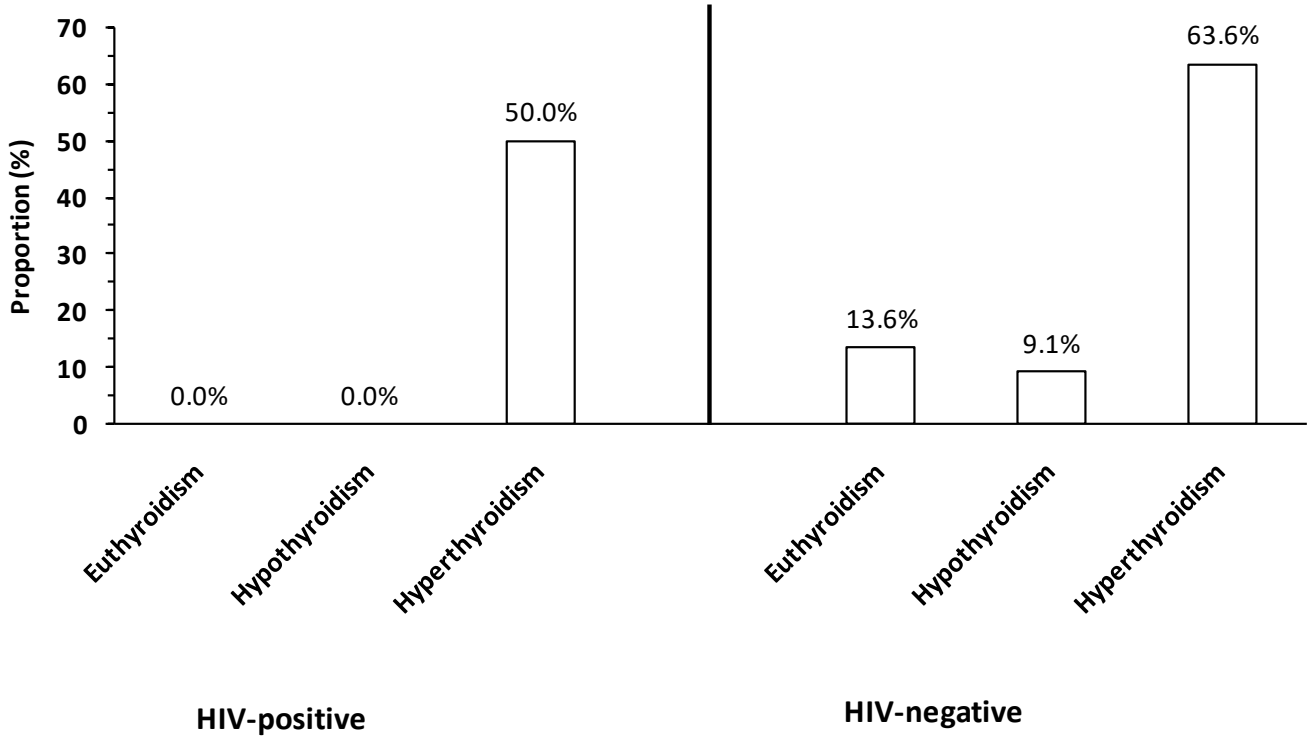

Figure 1
Prevalence of Thyroid Dysfunction among Pregnant Women

The figure presents the distribution of euthyroidism, hypothyroidism, and hyperthyroidism within each study group. Descriptive statistics in IBM SPSS statistics was used to calculate the proportions and percentages of participants within each thyroid status category.

This study findings revealed a high prevalence of thyroid dysfunction among HIV-positive pregnant women, consistent with prior reports (Beltran *et al.*, 2003; Adan *et al*., 2025). The predominance of hyperthyroidism suggests ART-induced immune reconstitution and HIV-driven metabolic changes as underlying mechanisms (Chen *et al.*, 2020). In comparison to a previous study on patients on HAART conducted in Meru by (Adan *et al.*, 2025), whose thyroid dysfunction prevalence was 51.9% among general population living with HIV infection.. The absence of hypothyroidism in HIV-positive women contrasts with findings in HIV-negative women, who displayed a broader spectrum of thyroid dysfunction. Likewise, in a study (Ge *et al*., 2022), that indicates that preeclampsia affects up to 8% of pregnancies worldwide, while pregnancy induced hypertension is the leading cause of maternal mortalities and morbidity, indicative of hyperthyroidism being its leading risk.

V. CONCLUSION & RECOMMENDATION

5.1 Conclusion

Thyroid dysfunction is highly prevalent among HIV-positive pregnant women in Kakamega, with hyperthyroidism predominating. Screening should be integrated into ANC and HIV programs to improve outcomes.

5.2 Recommendations

Incorporate thyroid screening into ANC for HIV-positive women. Develop Kenyan guidelines addressing HIV-thyroid interactions in pregnancy. Expand multicenter research to validate prevalence nationally. Train healthcare workers in thyroid management within HIV care.

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