International Journal of Health Science

ISSN 2764-0159 vol. 5, n. 32, 2025

ARTICLE 3

Acceptance date: 29/10/2025

PREVALENCE OF SUBCLINICAL HYPOTHYROIDISM IN PEDIATRIC PATIENTS WITH HIV: A CROSS-SECTIONAL STUDY IN GUATEMALA

Xavier Eduardo Anzueto Fernández
Department of Medicine, Faculty of Health Sciences, Rafael Landívar University, Guatemala
Department of Pediatrics, Roosevelt Hospital of Guatemala

Jessenia Sabrina Navas Castillo
Comprehensive Care Unit for HIV and Chronic Infections "Dr. Carlos Rodolfo Mejía Villatoro," Roosevelt Hospital, Guatemala.
Department of Molecular Biology, Faculty of Biology, Chemistry, and Pharmacy (FABIQ), Galileo University
Latin American Network for the Advancement of Science (REDALAC)

Julio Werner Juárez Lorenzana
Comprehensive Care Unit for HIV and Chronic Infections "Dr. Carlos Rodolfo Mejía Villatoro," Roosevelt Hospital, Guatemala.
Faculty of Medical Sciences, San Carlos University of Guatemala

Abstract: Introduction: Human immunodeficiency virus (HIV) infection leads to multi-organ involvement, including the endocrine system. Severe immunosuppression and advanced HIV disease may be associated with subclinical hypothyroidism (SH), which directly affects growth and development in children. **Methodology:** A descriptive, cross-sectional, retrospective study was conducted of 112 pediatric patients with HIV treated between 2008 and 2021 at the Comprehensive Care Unit for HIV and Chronic Infections at Roosevelt Hospital, analyzing epidemiological, clinical, immunological, virological, and hormon y data obtained from medical records. **Results:** 21.4% of patients had elevated TSH and 18.8% had subclinical hypothyroidism, of which 17.9% showed a need for levothyroxine supplementation. A higher prevalence of thyroid dysfunction was observed in patients with advanced clinical stages and high viral load. **Conclusion:** Thyroid dysfunction is common in children with HIV, highlighting the need for early detection and timely intervention to improve the clinical prognosis and quality of life of this population.

Keywords: HIV, Thyroid dysfunction, Subclinical hypothyroidism, Pediatrics, Guatemala

Introduction

HIV infection is a chronic systemic disease that progressively weakens the immune system, promoting opportunistic infections, neoplasms, and potentially death [1]. In pediatrics, it is a global problem with a sustained increase in cases. In 2020, an estimated 37.7 million people were living with HIV, including 1.7 million children under the age of 14; by 2022, the figure had risen to 39.0 million, with 1.5 million children affected and 53% of the total being women and girls [2]. In Guatemala, between 1984 and 2019, 37,556 cumulative cases of HIV were reported, with 5% corresponding to children under 15 years of age. Although the initial frequency of cases was increasing, reports have declined since 2008, which could reflect underreporting [3].

Since the beginning of the epidemic, and even before the introduction of highly active antiretroviral therapy (HAART), neuroendocrine changes have been observed at the central, adrenal, gonadal, and thyroid levels [4, 5, 6, 7]. In HIV-infected pediatric patients, although little studied, hypothyroidism—especially subclinical hypothyroidism (SH)—has been shown to be the most common thyroid dysfunction [6, 7]. SH is characterized by the absence of clinical symptoms but is ly associated with thyroid-stimulating hormone (TSH) levels above the upper limit of the reference range, together with a normal serum concentration of free thyroxine (FT4) [8]. This comorbidity may be associated with other complications, so it is crucial to correlate it with the clinical-immunological stage of HIV [1, 4].

Several studies have indicated that thyroid function abnormalities are more prevalent in HIV-infected patients than in the general population, although the exact cause of this dysfunction is still unclear. Among the proposed hypotheses are autoimmune processes in which autoantibodies attack multiple thyroid antigens. This effect, derived from the systemic action of HIV, manifests itself with the presence of antibodies such as antithyroid peroxidase or antithyroglobulin, which can lead to glandular hypofunction secondary to the

destruction of thyroid tissue, concurrent infections, damage associated with opportunistic infections, and adverse reactions to medications [9, 10, 11].

Previous studies have demonstrated an association between thyroid abnormalities and disease progression, including severe immunosuppression and high viral load; however, these findings are insufficient [3, 12]. Given that there is an inverse relationship between TSH levels and CD4+ T-cell counts, it has been suggested that severe immunosuppression and advanced HIV progression could increase the risk of developing HS, which directly impacts child growth by causing growth retardation and a decrease in the linear growth rate [4, 9, 13].

Considering the impact of thyroid disorders on the health of children with HIV, timely detection of this comorbidity is essential. Early initiation of thyroid hormone treatment is essential to prevent further deterioration in their growth and development, already compromised by the disease. The presence of antithyroid antibodies (anti-thyroid peroxidase or anti-thyroglobulin) can contribute to the destruction of thyroid tissue and, consequently, to the development of glandular hypofunction in HIV-positive pediatric patients [4, 10, 14].

The objective of this study was to determine the association between immunovirological parameters (viral load, CD4+ lymphocyte count) and clinical-immunological stage with the presence of subclinical hypothyroidism in HIV-infected children treated at the Dr. Carlos Rodolfo Mejía Villatoro Comprehensive Care Unit for HIV and Chronic Infections at Roosevelt Hospital in Guatemala. The results of this research could be considered for the implementation of protocols for early detection and timely management of thyroid dysfunction, which would contribute to optimizing the quality of life and overall development of these patients.

Methodology

Type of study and design

Descriptive, cross-sectional study.

Unit of analysis

Medical records of pediatric patients with HIV who underwent a baseline thyroid test (TSH and FT4) at the time of diagnosis during the period from 2008 to 2021.

Population and sample

Information from 112 medical records of pediatric patients infected with human immunodeficiency virus who were actively followed up in the pediatrics area of the Dr. Carlos Rodolfo Mejía Villatoro Comprehensive Care Unit for HIV and Chronic Infections at Roosevelt Hospital during the period from 2008 to 2021 was included.

Inclusion criteria

Medical records of pediatric patients (boys and girls) who underwent initial thyroid testing at the time of HIV diagnosis.

Exclusion criteria

Records of children with any other condition that alters thyroid function, such as chronic kidney disease, thyroid disease with a previous diagnosis of HIV, hepatitis B virus infection, hepatitis C, tuberculosis, autoimmune diseases, or drugs toxic to the thyroid (amiodarone, interferon, rifampicin).

Data collection instrument and process

A data collection form was used as an instrument, extracting the epidemiological variables of each patient from the medical records. HIV viral load and CD4+ T-cell count values were taken from the laboratory results registry of the Comprehensive Care Unit for HIV and Chronic Infections, while thyroid-stimulating hormone (TSH) and free thyroxine (FT4) were transcribed from the Roosevelt Hospital Endocrinology and Specialties Laboratory results registry, correlating with the patient's clinical condition described in the medical record. Subsequently, a digital database was created in Excel 2019, adapting the fields to the variables of interest, which were transcribed for subsequent classification and analysis.

Variables

The study variables cover epidemiological, clinical-immunological, virological, and endocrinological aspects. First, demographic variables such as sex and age were considered. In the context of HIV infection, clinical and immunological classification (CD4+ T lymphocytes) were evaluated, in addition to a combined variable that integrates both classifications. The HIV viral load was also analyzed. Finally, variables related to thyroid function were included: TSH and FT4 levels, and the resulting endocrinological diagnosis, as well as the need for levothyroxine supplementation in patients with subclinic .

Clinical and immunological classification according to age

Classification was based on immune stage, determined by age-adjusted CD4+ T-cell count, and clinical stage, according to the HIV classification established by the Centers for Disease Control and Prevention (CDC, USA, 1994). The immunological stage is categorized as: 1 (mild immunosuppression), 2 (moderate immunosuppression), and 3 (severe immunosuppression). The clinical stage is classified as: N (asymptomatic), A (mild signs and symptoms), B (moderate signs and symptoms), and C (severe signs and symptoms or AIDS-defining illnesses).

Data analysis

Data processing and analysis were performed using SPSS statistical software. Descriptive analysis included the calculation of absolute and relative frequencies for categorical variables such as sex, age, clinical-immunological classification, viral load, and presence of thyroid dysfunction. Confidence intervals (CI) of 95% were calculated for the proportions in order to estimate the accuracy of the estimates. The association between the diagnosis of thyroid dysfunction and clinical and immunological variables was evaluated using Pearson's chi-square test or Fisher's exact test, as appropriate.

Additionally, a logistic regression analysis was performed to identify possible factors associated with the presence of subclinical hypothyroidism in the pediatric population with HIV. Odds ratios (OR) with their respective 95% confidence intervals were calculated to determine the magnitude of the association. A p-value < 0.05 was considered statistically significant in all tests.

Possible biases

Selection bias. Since the sample was obtained from the medical records of pe-

diatric patients with HIV being followed up at a single center (Roosevelt Hospital), the results may not be fully representative of the pediatric population with HIV in other settings. In addition, the inclusion of only patients with thyroid tests available at the time of diagnosis may exclude those who were not evaluated, which could influence the interpretation of the relationship between HIV and thyroid dysfunction.

Information bias. Data collection from medical records may be subject to errors in the recording of clinical and laboratory information. Variability in the quality and completeness of records could affect the accuracy of the data analyzed, especially in relation to the patient's clinical condition and the correlation with TSH and FT4 values.

Limitations

One possible limitation of the study was the sample size, which could influence the generalization of the findings to a broader pediatric population with HIV. However, the results provide valuable information on thyroid function in this group. In addition, only FT4 and TSH were analyzed; nevertheless, these markers are essential for evaluating thyroid disorders.

The cross-sectional nature of the study limited the evaluation of temporal relationships between some variables, but it did allow for a detailed characterization of the study population. On the other hand, the reduction in the number of pediatric visits due to the SARS-CoV-2 pandemic may have affected case detection, although it did not compromise the relevance of the results obtained.

Ethical considerations

In this research, both the population and the units of analysis consisted of clinical records, so there were no risk implications for patients, as no invasive procedures were performed. Access to the research instrument was restricted to the researcher only, ensuring confidentiality throughout the process. The research protocol was reviewed and approved by the Teaching and Research Department of Roosevelt Hospital, registered on June 27, 2022, in Act 680, Point 3.

Results

Epidemiological, clinical, immunological, virological, and hormonal data obtained from the clinical records of 112 pediatric patients with HIV treated between 2008 and 2021 at the Comprehensive Care Unit for HIV and Chronic Infections at Roosevelt Hospital were analyzed.

Characteristic	f	%	95% CI
Sex			
Female	63	56.3	46.8% - 65.2%
Male	49	43.8	34.8% - 53.2%
Age			
Under 3	44	39.3	29.9% - 48.1%
3 to 5 years	20	17.9	10.9% - 25.2%
More than 5 years	48	42.9	33.8% - 52.2

Table 1. Epidemiological characteristics of pediatric patients infected with HIV

Classification	f	%	95% CI
Clinical stage			
N – asymptomatic	13	11.6	5.9% - 18.1%
A – mild	15	13.4	6.7% - 19.3%

Classification	f	%	95% CI
B – moderate	34	30.4	21.5% - 38.5%
C – severe	50	44.6	35.7% - 54.3%
Immunological stage			
1: no immunosuppression	77	68.8	60.4% - 77.6%
2: moderate immunosuppression	17	15.2	8.4% - 21.6
3: severe immunosuppression	18	16.1	9.2% - 22.8%

Table 2. Clinical-immunological classification of pediatric infection

Viral load	f	%	95% CI
Below detection limit (<40 cp/mL)	7	6.3	1.6% - 10.4
< 1,000 cp/mL	11	9.8	4.4% - 15.6%
1,000 – 10,000 cp/mL	15	13.4	6.7% - 19.3%
> 10,000 cp/ml	79	70.5	62.6% - 79.4%

Table 3. Initial HIV viral load at the time of the study in pediatric patients infected with HIV

Stage	f	%	95% CI
N1	11	9.8	4.4% - 15.6%
N2	0	0.0	0.0% - 0.0%
N3	2	1.8	0.6% - 4.6%
A1	6	5.4	0.9% - 9.1%
A2	7	6.3	1.6% - 10.4%
A3	2	1.8	0.6% - 4.6%
B1	9	8.0	2.9% - 13.1%
B2	15	13.4	6.7% - 19.3%
B3	10	8.9	3.7% - 14.3%
C1	7	6.3	1.6% - 10.4%
C2	13	11.6	5.9% - 18.1%
C3	30	26.8	18.7% - 35.3%

Table 4. Combined clinical-immunological classification in pediatric patients with HIV

Level	f	%	95% CI
TSH Decreased	1	0.9	0.8% - 2.9%
Normal TSH	87	77.7%	70.3% - 85.7%
Elevated TSH	24	21.4	13.4% - 28.6%
Decreased FT4	6	5.4	0.9% - 9.0%
Normal FT4	105	93.8	89.6% - 98.4%
Elevated FT4	1	0.9	0.8% - 2.9%

TSH: Thyroid-Stimulating Hormone

FT4: Free Thyroxine

Table 5. Baseline thyroid hormone levels at the time of HIV diagnosis

Diagnosis	f	%	95% CI
Euthyroid	89	79.5	71.4% - 86.6%
Subclinical hypothyroidism	21	18.8	11.7% - 26.3%
Hyperthyroidism	1	0.9	0.9% - 2.9%
Sick euthyroid syndrome	1	0.9	0.9% - 2.9

Table 6. Classification of thyroid dysfunction in pediatric patients with HIV

Need for supplementation	f	%	95% CI
Yes	20	17.9	10.9% - 25.2%
No	92	82.1	74.9% - 89.2%

Table 7. Need for levothyroxine supplementation in HIV-positive patients with subclinical hypothyroidism

In the study population, 56.3% of patients were female, and 42.9% were over 5 years of age. Regarding clinical classification, 44.6% were in clinical stage C (severe), while 16.1% had severe immunosuppression (immunological stage 3).

Category	Subclinical hypothyroidism (%)	Euthyroid (%)	OR	p	95% CI
Clinical stage					
N - Asymptomatic	15.4	19.2	0.77	0.742	0.157 - 3.745
A - Mild	20.0	18.6	1.10	0.894	0.280 - 4.296
B - Moderate	17.6	19.2	0.90	0.844	0.316 - 2.562
C - Serious	20.0	17.7	1.16	0.761	0.448 - 3.001
Immunological stage					
1: No immunosuppression	17.2	19.4	0.89	0.798	0.280 - 2.663
2: Moderate immunosuppression	12.9	21.4	0.54	0.317	0.164 - 1.795
3: Severe immunosuppression	24.4	15.0	1.83	0.240	0.669 - 4.994
Clinical/immunological stage ratio					
N1	18.2	18.8	0.96	0.96	0.191 - 4.805
N2	0.0	0.0	-	-	
N3	0.0	19.1	0.83	0.907	0.039 - 17.983
A1	33.3	17.9	2.29	0.359	0.391 - 13.421
A2	14.3	19.0	0.71	0.756	0.081 - 6.219
A3	0.0	19.1	0.83	0.907	0.039 - 17.983
B1	22.2	18.4	1.26	0.781	0.243 - 6.567
B2	0.0	21.6	0.11	0.138	0.007 - 1.997
B3	40.0	16.7	3.33	0.085	0.849 - 13.094
B3 men	8.3	8.1	1.03	0.980	0.097 - 10.946
B3 women*	33.3	5.6	8.5	0.021	1.391 - 51.95
C1	0.0	20.0	0.26	0.366	0.014 - 4.770
C2	23.1	18.2	1.35	0.672	0.337 - 5.407
C3	23.3	17.1	1.48	0.454	0.531 - 4.112
HIV viral load					
CV > 1000 cp/ml	19.1	16.7	1.18	0.805	0.310 - 4.531
CD4+ T lymphocytes					
CD4+ T cells < 200 ul/mm³	28.6	17.6	1.87	0.258	0.631 - 5.575
T CD4+ < 500 ul/mm³	47.6	46.6	1.06	0.903	0.410 - 2.743

*Statistically significant results ($p < 0.05$)

Table 8. Association between thyroid disorder diagnosis and indicators of human immunodeficiency virus infection status in the pediatric population

Likewise, 70.5% of patients had a viral load greater than 10,000 cp/ml. Distribution according to clinical-immunological stage showed that most patients were in stage C3 (26.8%) and B2 (13.4%). With regard to thyroid function, TSH and FT4 levels were normal in 77.7% and 93.8% of cases, respectively.

Seventy-nine point five percent of patients were euthyroid, while 18.8% had subclinical hypothyroidism. Of the 23 patients with thyroid dysfunction, 20 required levothyroxine supplementation, representing 17.9% of the total population analyzed. When analyzing the distribution of subclinical hypothyroidism (SH), it was observed that 20.0% of patients with SH were in clinical stage C, compared to 17.7% of those without thyroid disease in the same stage, with no statistically significant differences.

Regarding the immunological stage, 24.4% of patients with SH were in stage 3, compared to 15.0% of euthyroid patients. The clinical-immunological stages most associated with SH included A1, B1, B3, C2, and C3. A significant association was identified in stage B3 in women, who had a prevalence of subclinical hypothyroidism of 33.3% (OR = 8.50; p = 0.021), compared with 8.3% in men.

Discussion

Human immunodeficiency virus (HIV) infection can lead to multiple organ involvement, including the endocrine system. This is due to the direct impact of the virus on the immune system, which causes an alteration in endocrine function. [15, 16]

The findings in this study, which show a prevalence of subclinical hypothyroidism in 18.8% of the pediatric population with HIV, are higher than those reported in international studies. For example, Beltran et al. described a prevalence of 8.1% in HIV-infected adults [5], while Merenich et al. reported similar figures prior to the introduction of ART [17]. This difference could be attributed to the pediatric population profile, the type of antiretrovirals used, and the conditions of clinical follow-up in resource-limited settings such as Guatemala.

Regarding the association between thyroid function and immune status, the results are consistent with the observations of Jain et al., who reported a correlation between thyroid abnormalities and advanced immune progression (measured by CD4+ T-cell counts) [18]. Thongam et al. demonstrated a significant positive correlation between CD4+ T-cell counts and TSH levels [9], suggesting that the likelihood of thyroid dysfunction increases as the count of these cells decreases or the disease progresses. Complementarily, a study conducted in India found a direct correlation between CD4+ cell count and free T3 ($r = 0.357$, $p < 0.05$) and free T4 ($r = 0.650$, $p < 0.05$), as well as an inverse correlation between CD4+ cell count and serum TSH levels ($r = 0.470$, $p < 0.050$) [19].

However, a study conducted in Iran did not identify any significant relationship between HS, HIV infection, and CD4+ cell count [20], which is largely consistent with our analysis, in which no statistically significant association was observed, although a trend toward a higher frequency of thyroid dysfunction was identified in advanced clinical stages of HIV, particularly in stages B3, C2, and C3.

On the other hand, the identification of a case with sick euthyroid syndrome coin-

cides with that described by Chiarelli et al., who reported this entity in children with perinatal HIV, especially in phases of severe immunosuppression [21]. This alteration, considered an adaptive response to systemic stress, was also described by Lo Presti et al. as a marker of poor prognosis in the context of severe infections [22].

In terms of management, the need for levothyroxine supplementation in 17.9% of patients in this cohort is in line with the literature, which emphasizes the importance of pharmacological intervention in cases of hypothyroidism, even subclinical, to avoid negative effects on child growth and development [23,24].

Likewise, the finding of a higher prevalence of HS in women, especially in stage B3 (33.3%), provides new data that, although not widely described in other studies, could be related to hormonal, genetic, or differential autoimmune factors in the female population infected with HIV, a topic that warrants further study.

The findings of this study highlight the need to establish strategies aimed at early detection and periodic monitoring of thyroid dysfunction in children living with HIV, with special emphasis on those with subclinical hypothyroidism. These results support the implementation of standardized institutional protocols for regular hormone assessment and the definition of precise clinical criteria for indicating levothyroxine supplementation in selected cases.

Similarly, it highlights the importance of promoting future longitudinal research to analyze the evolution of thyroid disorders and their impact on child development, as well as their possible interaction with immunovirological control. Finally, exploring related environmental, genetic, and immunological factors could provide key insights for identifying additional determinants of thyroid dysfunction in this vulnerable population, thereby strengthening comprehensive interventions and improving the quality of life of pediatric patients living with HIV.

Conclusion

This study shows a high prevalence of thyroid dysfunction, mainly subclinical hypothyroidism, in pediatric patients with HIV treated at the Comprehensive Care Unit of Roosevelt Hospital. Most of these patients required levothyroxine supplementation, highlighting the need for ongoing endocrinological monitoring. The results underscore the importance of integrating thyroid evaluation into the clinical management of this vulnerable population.

Acknowledgments

We would like to thank the staff of the Comprehensive Care Unit for HIV and Chronic Infections, as well as Gretel Lemus and Pablo Tzorin of the Endocrinology and Specialties Laboratory at Roosevelt Hospital, for their support in the development of this study. We also thank the Department of Medicine of the Faculty of Health Sciences of Rafael Landívar University for their support in the completion of the undergraduate thesis: "Prevalence of hypothyroidism in pediatric patients with HIV from 2008 to 2021 in Guatemala," from which this article is derived.

References

1. Maldonado Lara E. Disfunción tiroidea y VIH: Prevalencia, correlación clínica e inmunológica en una cohorte de pacientes seropositivos [Internet]. 1ª ed. Bogotá: Universidad del Rosario; 2018 [citado 2021 mar 1]. Disponible en: https://repository.urosario.edu.co/bitstream/handle/10336/18121/MaldonadoLara-Eliana-2018.pdf?sequence=1&isAllowed=y

2. ONUSIDA. Hoja informativa — últimas estadísticas sobre el estado de la epidemia de sida [Internet]. 2020 [citado 2021 ago 16]. Disponible en: https://www.unaids.org/es/resources/fact-sheet

3. ONUSIDA. Actualización mundial sobre el sida 2017; julio 2017 [Internet]. ONUSIDA; 2017 [citado 2021 mar]. Disponible en: http://aidsinfo.unaids.org

4. Juárez J, Rodríguez D, Gramajo A, Velasco R, Reynoso L. Incidencia de disfunción tiroidea en niños VIH positivos. Rev Fac Med Univ Francisco Marroquín [Internet]. 2009 [citado 2021 mar 1];(1137-2834):10–13. Disponible en: https://medicina.ufm.edu/images/9/99/2009-01_06.pdf

5. Beltran S, Lescure FX, Desailloud R, Douadi Y, Smail A, El Esper I, et al. Aumento de la prevalencia de hipotiroidismo entre los pacientes infectados por el VIH: una necesidad de detección. Clin Infect Dis. 2003;37(4):579–83. https://doi.org/10.1086/376626

6. Sellmeyer D, Grunfeld C. Alteraciones endocrinas y metabólicas en la infección por el VIH y el síndrome de inmunodeficiencia adquirida. Endocr Rev [Internet]. 1996 [citado 2021 abr 16];17(5):518–32. Disponible en: https://academic.oup.com/edrv/article/17/5/518/2548601 doi:10.1210/edrv-17-5-518

7. Khalsa J, Genser S, Coates P, Francis H. Intervenciones para las complicaciones metabólicas y endocrinas del VIH/sida y el uso de drogas ilícitas. Clin Infect Dis [Internet]. 2003 [citado 2021 abr 16];37(Suppl 2):S37–42. Disponible en: https://academic.oup.com/cid/article/37/Supplement_2/S37/336113 doi:10.1086/375890

8. Salerno M, Capalbo D, Cerbone M, De Luca F. Hipotiroidismo subclínico en la infancia: conocimiento actual y cuestiones abiertas. Nat Rev Endocrinol. 2016;12(12):734–46. doi:10.1038/nrendo.2016.100

9. Thongam S, Keithelakpam S, Singh TY, Singh RL, Singh AM, Ranabir S, et al. Disfunción tiroidea en niños infectados por el VIH y su correlación con el recuento de linfocitos T CD4(+). Indian J Endocrinol Metab. 2015;19(2):272–6. doi:10.4103/2230-8210.149321 PMID:25729691

10. Mayer KH, Hoffmann CJ, Brown TT. Anomalías de la función tiroidea en pacientes infectados por el VIH. Clin Infect Dis. 2007;45(4):488–94. doi:10.1086/519978

11. Vigano A, Riboni S, Bianchi R, Cafarelli L, Vago T, Manzoni P, et al. Disfunción tiroidea en niños tratados con antirretrovirales. Pediatr Infect Dis J [Internet]. 2004 [citado 2021 abr 16];23(3):235–9. Disponible en: https://journals.lww.com/pidj/Abstract/2004/03000/Thyroid_dysfunction_in_antiretroviral_treated.12.aspx doi:10.1097/01.inf.0000114903.05472.e4

12. John B, Gupta R, Pandey A. Anomalías endocrinológicas y deterioro del crecimiento en niños infectados por el VIH. J Pediatr Infect Dis. 2018;13(1):63–9. doi:10.1055/s-0037-1617414

13. Akinsete A, Oyenusi E, Odugbemi B, Odugbemi T, Temiye E. Espectro de anomalías tiroideas entre los niños que viven con el VIH en Lagos, Nigeria. Thyroid Res. 2019;2019:1096739. doi:10.1155/2019/1096739

14. Fundaró C, Olivieri A, Rendeli C, Genovese O, Martino A, D'Archivio M, et al. Aparición de autoanticuerpos antitiroideos en niños infectados verticalmente con VIH-1. J Pediatr Endocrinol Metab [Internet]. 1998 [citado 2021 abr 16];11(6):745–52. Disponible en: https://europepmc.org/article/med/9829230 doi:10.1515/jpem.1998.11.6.745

15. Raffi F, Brisseau JM, Planchon B. Función endocrina en 98 pacientes infectados por el VIH: un estudio prospectivo. AIDS. 1991;5:729–33.

16. Dobs AS, Dempsey MA, Ladenson PW. Trastornos endocrinos en hombres infectados con el VIH. Am J Med. 1988;84:611–6.

17. Grappin M, Piroth L, Verges B, Sgro C, Mack G, Buisson M, et al. Aumento de la prevalencia de hipotiroidismo subclínico en pacientes con VIH tratados con TAR altamente activa. AIDS. 2000;14(8):1070–2. doi:10.1097/00002030-200005260-00026 PMID:10853996

18. Jain G, Devpura G, Gupta BS. Anomalías en las pruebas de función tiroidea como marcador sustituto del avance de la infección por VIH en adultos infectados. J Assoc Physicians India. 2009;57:508–10.

19. Afhami S, Haghpanah V, Heshmat R, Rasoulinejad M, Izadi M, Lashkari A, et al. Evaluación de los factores implicados en el desarrollo de hipotiroidismo en pacientes infectados por el VIH: un estudio de casos y controles. Infection. 2007;35(5):334–8. doi:10.1007/s15010-007-6163-3 PMID:17885731

20. Merenich JA, McDermott MT, Asp AA, Harrison SM, Kidd GS. Evidencia de compromiso endocrino temprano en la infección por VIH. J Clin Endocrinol Metab. 1990;70(3):566–71. doi:10.1210/jcem-70-3-566

21. Güemes M, Corredor Andrés B, Muñoz Calvo MT. Patología tiroidea en la infancia y la adolescencia. Pediatr Integral. 2020;XXIV(5):248–57. Disponible en: https://www.pediatriaintegral.es/publicacion-2020-07/patologia-tiroidea-en-la-infancia-y-la-adolescencia/

22. Lo Presti JS, Fried JC, Spencer CA. Alteraciones únicas de los índices de hormonas tiroideas en el SIDA. Ann Intern Med. 1989;110:970–5.

23. Varthakavi PK. Disfunción tiroidea en el VIH-SIDA. J Assoc Physicians India. 2009;57(7):503–4.

24. Schwartz LJ, St Louis Y, Wu R, Wiznia A, Rubinstein A, Saenger P. Endocrine function in children with human immunodeficiency virus infection. Am J Dis Child. 1991 Mar;145(3):330-3. doi: 10.1001/archpedi.1991.02160030100030. PMID: 2003485.