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Prevalence of Adrenocortical Insufficiency in Treatment-Naïve HIV Patients in Maiduguri: A Cross-Sectional Study

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Abstract **Original Research Article**

Background: Human immunodeficiency virus (HIV) infection is often associated with various endocrine disorders, including adrenal dysfunction, particularly adrenocortical insufficiency (AI), which represents a significant yet frequently overlooked complication in HIV-infected individuals, particularly within resource-constrained healthcare environments. The condition remains substantially underdiagnosed despite its potential for severe clinical consequences. While highly active antiretroviral therapy (HAART) is known to improve immune function and reduce comorbidities, its effect on adrenal function, particularly in treatment-naïve HIV-infected patients, requires further investigation. ***Objective:*** This study aimed to determine the prevalence of adrenocortical insufficiency (AI) in treatment-naïve HIV-infected patients in Maiduguri. ***Methods:*** This cross-sectional study involved newly diagnosed, treatment-naïve HIV patients and apparently healthy individuals. Adults aged ≥18 years were enrolled, with 138 stable HIV patients and 132 healthy subjects completing the study. Data were collected using a standardized sheet. Serum cortisol levels were measured at 0, 30, and 60 minutes after a 1µg ACTH (adrenocorticotrophic hormone) stimulation test. Normal cut-off reference values for serum cortisol were derived from the healthy subjects. AI was defined as a peak serum cortisol level of < 504 nmol/L at 30 or 60 minutes after 1µg ACTH stimulation and an increment of < 124 nmol/L. Data were analyzed using Statistical Package for Social Sciences (SPSS) version 23.0, with statistical significance set at $P < 0.05$. ***Results:*** A total of 270 participants completed the study, and data from 220 subjects (129 HIV-positive, 91 healthy subjects) were analyzed. Among HIV patients, 58 (42.0%) were males and 80 (58.0%) were females. The mean age of HIV patients was 36.07±8.87 years, significantly higher than healthy subjects (31.19±11.18 years, $p = 0.001$). The median basal (0 min) cortisol levels and cortisol levels at 30 and 60 minutes after ACTH stimulation were significantly lower in HIV patients than in healthy subjects ($p < 0.001$ for all time points). The prevalence of adrenocortical insufficiency among newly diagnosed, treatment-naïve HIV patients was 33.3% after ACTH stimulation and 52.7% when assessed using basal cortisol concentrations. While clinical features like weight loss and weakness were common in HIV patients, there was no statistically significant difference in clinical features between HIV patients with AI and those with normal adrenal function. ***Conclusion:*** Adrenocortical insufficiency is relatively common among newly diagnosed treatment-naïve HIV-infected patients in Maiduguri, affecting approximately one-third of them. The classical clinical features of AI are often absent or overlap with symptoms of advanced HIV infection, necessitating a high index of suspicion and biochemical diagnosis for early intervention.

Keywords: Adrenocortical-insufficiency acquired immunodeficiency syndrome, highly active antiretroviral therapy, human immunodeficiency virus, HIV/AIDS, low-dose short synacthen test, low dose short synacthen test.

INTRODUCTION

Human immunodeficiency virus (HIV) infection is a chronic condition that can lead to acquired immunodeficiency syndrome (AIDS), affecting various physiological systems, including the endocrine system.[1] Among the myriads of endocrine disorders associated with HIV, dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis is particularly common .[1–3] This dysfunction can arise from direct viral effects,

opportunistic infections, malignancies infiltrating endocrine glands, immune activation, hypercytokinemia, or side effects of antiretroviral therapy (ART).[1,4]

Adrenal gland involvement has been histologically documented in up to two-thirds of AIDS patients post-mortem.[1,4,5] However, clinical adrenal insufficiency (AI) is often underdiagnosed because its symptoms tend to manifest only after significant gland destruction (80-90%).[1,4,5] Furthermore, the non-specific nature of AI symptoms such as fatigue, weight loss, and gastrointestinal issues often overlaps with features of advanced HIV infection, making clinical differentiation challenging without biochemical confirmation.[1,6] Despite this diagnostic difficulty, AI is associated with significant morbidity and mortality, emphasizing the importance of timely diagnosis and treatment .[3]

The global prevalence of HIV remains substantial, with Sub-Saharan Africa bearing a disproportionately high burden.[7,8] Nigeria, specifically, reported an HIV prevalence of approximately 3.2% among adults in 2016.[8] Previous studies in Nigeria have reported varying prevalences of adrenal dysfunction in HIV patients. For instance, a study in Lagos, Southwest Nigeria, found a prevalence of adrenocortical insufficiency of 34.8% among treatment-naïve HIV patients.[9] Another study in Northwest Nigeria reported a total adrenal dysfunction prevalence of 13.8% in HIV patients, with hypofunction being more common than hyperfunction,[10] and a prevalence of 27% was reported in South Africa, highlighting the regional significance of this complication.[11] While these studies provide insights into adrenal dysfunction in Nigerian HIV patients and Sub-Saharan Africa, there remains a gap in published data regarding the prevalence of AI, specifically in treatment-naïve HIV patients in Maiduguri, Northeast Nigeria.

The low-dose short synacthen test has emerged as a more sensitive diagnostic tool compared to standard-dose ACTH stimulation testing, particularly for detecting subtle adrenal dysfunction in early disease stages.[12,13] This enhanced sensitivity is particularly relevant in resource-limited settings where early detection and intervention are crucial for preventing adverse outcomes.

This study aims to address these gaps by evaluating the prevalence of adrenocortical insufficiency in newly diagnosed, treatment-naïve HIV patients in Maiduguri, Nigeria. This foundational assessment will provide crucial local data for clinical practice and further research in the region.

METHODS

Study Design and Site This was a cross-sectional study, focusing on baseline measurements. The study was conducted in Maiduguri, Borno State, Northeast Nigeria, at the University of Maiduguri Teaching Hospital (UMTH) and State Specialist Hospital Maiduguri (SSHM). These are major referral centers serving Borno and neighboring states, including parts of Cameroun, Niger, and Chad.

Study Population and Sample Size The target population included newly diagnosed, treatment-naïve HIV-positive adults aged ≥18 years attending the infectious disease outpatient clinics at UMTH and SSHM. A control group of apparently healthy HIV-negative individuals, aged ≥18 years, was recruited from voluntary counseling and testing (VCT) centers and blood donation units at UMTH, as well as general volunteers.

The sample size was calculated using a formula for proportion: $n = Z^2pq/d^2$.[14] Based on a previously reported prevalence of adrenal dysfunction in newly diagnosed treatment-naïve HIV patients of 10.0%,[10] a 95% confidence interval (Z=1.96), and a desired accuracy of 0.05, the calculated sample size was 138. Allowing a 10% attrition rate, 153 HIV-positive patients were targeted, along with an equal number (153) of healthy control individuals. Ultimately, 138 HIV patients and 132 healthy subjects completed the study, with data from 129 HIV-positive participants and 91 healthy subjects analyzed after excluding outliers and those with equivocal ACTH responses.

Eligibility Criteria

Inclusion Criteria for HIV Patients

Newly diagnosed, treatment-naïve HIV patients (less than 1 year of diagnosis), male or female subjects aged ≥18 years, and provided informed consent to participate.

Exclusion Criteria for HIV Patients

Patients currently on any form of antiretroviral therapy, patients on steroid medications, acutely ill patients requiring hospital admission, pregnant women, patients with concomitant diabetes mellitus, individuals <18 years, and subjects who declined consent.

Inclusion Criteria for Healthy Subjects

Healthy normal adults, HIV negative, male or female subjects aged ≥18 years, consented to participate in the study.

Exclusion Criteria for Healthy Subjects

HIV positive, individuals on steroid or ketoconazole medications, pregnant women, individuals younger than 18 years, and those who declined consent.

Data Collection Procedures

A standardized data collection sheet was used to gather comprehensive information, including biodata, medical history (e.g., weakness, fatigue, weight loss, nausea, vomiting, hyperpigmentation), and history of glucocorticoid or antiretroviral therapy. Physical

examination included blood pressure measurements (supine and erect), and anthropometric measurements (weight and height).

Clinical and Laboratory Procedures

Participants arrived at the chemical pathology laboratory of UMTH on the assigned day, 60 minutes before the ACTH test, following an overnight fast of 8-10 hours. A 21-G cannula was inserted into a cubital vein and kept patent with heparinized saline, with subjects resting for 30 minutes before sample collection.

The 1µg low dose short synacthen test (LDSST) was performed for screening of adrenal dysfunction. A 1ml (250µg) vial of ACTH (synacthen) was diluted with 249ml of 0.9% normal saline to achieve a concentration of 1µg/ml. Blood samples were collected for basal cortisol levels (0 minutes), immediately before intravenous administration of 1µg ACTH.[9] Subsequent blood samples for cortisol were drawn at 30- and 60-minutes post-ACTH administration. Samples were centrifuged, plasma/serum separated, and stored at -20°C until assay.

Serum cortisol levels were determined using an enzyme-linked immunosorbent assay (ELISA) kit (Calbiotech Inc., CA, USA, Cat#: CO103S).[15] All participants completed the study with no adverse effect.

Diagnostic Criteria

Adrenal Insufficiency (AI):

In this study, AI was defined based on derived cut-off values from healthy subjects. At basal serum cortisol level, AI was defined as <207.56 nmol/L. Following 1µg ACTH stimulation, AI was defined as a peak serum cortisol level at 30 or 60 minutes < 504 nmol/L *plus* an increment from basal to stimulated cortisol level < 124 nmol/L. A normal response was defined as peak serum cortisol ≥504 nmol/L *and* increment ≥124 nmol/L

Ethical Considerations

The study protocol received approval from the ethics and research committee of the University of Maiduguri Teaching Hospital. All participants received detailed information about the study and provided written informed consent before enrollment. Confidentiality of participant data was maintained throughout the study using unique codes instead of names, and the research adhered to the Helsinki Declaration.

Statistical Analysis

Statistical analysis was performed using SPSS version 23.0. Summary descriptive statistics were reported as mean ± standard deviation for normally distributed continuous data, and median (interquartile range) for skewed continuous data. Comparisons between continuous data were made using independent t-tests or Mann-Whitney U tests, while categorical data were compared using chi-square tests and expressed as proportions. Statistical significance was set at $P < 0.05$.

RESULTS

Participant Flow

A total of 306 participants were initially recruited: 153 individuals with HIV infection and 153 healthy subjects. Of these, 270 participants completed the study (138 HIV patients and 132 healthy subjects), yielding a completion rate of 88.2% and an attrition rate of 11.8%. For the final analysis, data from 129 HIV-positive participants and 91 healthy subjects were analyzed, after excluding outliers and those with equivocal ACTH responses.

Baseline Characteristics of Study Participants

Among the 138 HIV-infected participants, 58 (42.0%) were males and 80 (58.0%) were females, resulting in a male-to-female ratio of 1:1.4. In the healthy subject group, there were 72 (54.5%) males and 60 (45.5%) females, with a male-to-female ratio of 1:0.8. The mean age of HIV patients (36.07±8.87 years) was significantly higher than that of healthy subjects (31.91±11.18 years, p = 0.001).

Table 1: Baseline Characteristics of Study Participants

Variables	Mean ±SD		Md	95% CI	P-value
	HIV Positive N=138	Healthy Subjects N=132			
Gender*	n (%)	n (%)			0.004
Male	58 (42.0)	72 (54.5)			
Female	80 (58.0)	60 (45.5)			
M: F Ratio	1:1.4	1:0.8			
Age	36.07±8.87	31.91±11.18	4.16	1.74 to 6.59	0.001
Anthropometry					
Weight (Kg)	58.03±11.63	66.68±11.47	-8.650	-11.77 to -5.53	**<0.001**
BMI (Kg/m^2)	20.97±4.14	23.71±4.69	-2.735	-3.92 to -1.55	**<0.001**

**χ^2 =4.234, df = 1, p = 0.040; CI, confidence interval; M±SD, mean ± standard deviation; Md, mean difference; M: F, male-to-female ratio.*

Baseline Serum Cortisol Levels in HIV-Positive Participants and Healthy Subjects

The median (interquartile range, IQR) basal cortisol level (0-minute) in HIV-positive patients was 200.27 nmol/L (134.80-273.38), which was significantly lower than in healthy subjects [310.62 nmol/L (258.48-393.10), $p < 0.001$]. Similarly, post-ACTH stimulation cortisol levels at 30 and 60 minutes, and their corresponding increments from basal, were all significantly lower in HIV patients compared to healthy subjects ($p < 0.001$ for all). Maximal stimulation in HIV patients was observed at 30 minutes post-ACTH.

Table 2: Serum Cortisol Levels in HIV-Positive Participants and Healthy Subjects

Time	Cortisol Level [Median (IQR)] (nmol/L)			
	HIV Positive	Healthy Subjects	U test	P-value
0-minutes	200.27 (134.07 – 273.38)	310.62 (258.48 – 393.10)	2495.0	**<0.001**
30-minutes	422.34 (263.72 – 514.07)	604.96 (531.58 – 683.58)	1616.0	**<0.001**
Increment at 30-min	156.69 (98.76 – 282.35)	293.24 (224.27 – 345.65)	2744.0	**<0.001**
60-minutes	274.48 (195.31 – 468.00)	498.48 (467.86 – 540.96)	2032.5	**<0.001**
Increment at 60-min	57.10 (32.28 – 129.38)	175.72 (112.00 – 248.27)	2489.5	**<0.001**

At 0-minute, serum cortisol levels at basal before stimulation with ACTH (adrenocortrophic hormone); At 30-minute, serum cortisol levels 30-minute post-ACTH stimulation; Increment at 30-min, an increment from basal to 30-minute post-ACTH stimulation; At 60-minute, serum cortisol levels 60 minutes post ACTH stimulation; Increment at 60-min, an increment from basal to 60-minute post-ACTH stimulation; HIV, human immunodeficiency virus; IQR, interquartile range; U test, Mann-Whitney U test.

Frequency of Adrenocortical Status of HIV-Positive Patients

Applying the derived cut-off values, the frequency of AI among newly diagnosed treatment-naïve HIV patients (N=129) was determined. At basal cortisol level, 68 (52.7%) patients exhibited AI, while 61 (47.3%) had a normal response. After 1µg ACTH stimulation, the frequency of AI decreased to 43 (33.3%), with 86 (66.7%) showing a normal adrenal response.

Table 3: Prevalence of Adrenal Insufficiency at 0-, 30- and 60-Minutes Criteria

Diagnostic Criteria	AI n (%)	Normal Response n (%)	Total n (%)
Basal Cortisol Level	68 (52.7)	61 (47.3)	129 (100.0)
Post-Stimulation Cortisol at 30 minutes	43 (33.3)	86 (66.7)	129 (100.0)
Post-stimulation Cortisol at 60 minutes	81 (62.8)	48 (37.2)	129 (100.0)

AI, adrenal insufficiency.

Clinical Features of Adrenocortical Insufficiency among HIV Patients

The study showed that there was no statistically significant difference in the distribution of clinical features between HIV patients with AI and those with normal adrenal function, as shown in Table 4.0.

Table 4: Clinical Features of Adrenocortical Insufficiency among HIV Patients

Clinical Features	Adrenal Statues				
	All	AI	No AI	X^2	P-value
N	**129**	**43**	**86**		
Weight loss	74 (57.4)	24 (55.8)	50 (58.1)	0.004	0.950
Weakness	61 (47.3)	22 (51.2)	39 (45.3)	0.190	0.663
Vomiting	9 (7.0)	4 (9.3)	5 (5.8)	0.134	0.714
Diarrhoea	13 (10.1)	2 (4.7)	11 (12.8)	1.294	0.255
Nausea	7 (5.4)	2 (4.7)	5 (5.8)	0.000	0.999
Hyperpigmentation	14 (10.9)	6 (14.3)	8 (9.3)	0.250	0.617
Postural hypotension	6 (4.7)	2 (4.7)	4 (4.7)	0.000	0.999

X^2, Chi-Square test. AI, adrenocortical insufficiency.

DISCUSSION

This study provides important insights into the prevalence of adrenocortical insufficiency in newly diagnosed, treatment-naïve HIV patients in Maiduguri, Northeast Nigeria.

The reliability of the study's findings is supported by the satisfactory performance of the laboratory assays for serum cortisol (using ELISA).[16,17] The use of the 1µg low dose short synacthen test (LDSST) for ACTH stimulation is a strength, as it is considered more physiological and sensitive for detecting subtle adrenal disturbances

compared to the standard 250µg test, which can lead to overstimulation and mask AI. The study's participant completion rate of 88.2% is acceptable, contributing to the robustness of the data.[18,19]

The derived cut-off reference values for serum cortisol in healthy subjects in this study (basal ≥207.56 nmol/L; post-stimulation peak ≥504 nmol/L and increment ≥124 nmol/L) align closely with recommendations from the Endocrine Society Clinical Practice Guidelines.[20] The inclusion of an increment criterion further enhances the diagnostic sensitivity and specificity.[9,21] It is acknowledged that newer assay kits may suggest lower cut-off values due to reduced cross-reactivity,[22] but the current study adhered to established guidelines for clinical practice.[20]

Consistent with other studies, the serum cortisol levels at all measured time points were significantly lower in HIV-infected patients compared to healthy individuals.[9,23] This suggests a generalized dysregulation of cortisol in HIV infection.

The prevalence of AI among newly diagnosed treatment-naïve HIV patients in this study was 33.3% after ACTH stimulation. This finding is comparable to that reported by Odeniyi *et al.,* in Lagos, Nigeria, who found a prevalence of 34.8%.[9] However, it is higher than the 16.3% post-stimulation prevalence reported by Akase *et al.,* in Kano, Nigeria,[24] and other international studies, which reported prevalences ranging from 14.06% to 27%.[10,11,25] The observed variations in AI prevalence across studies can be attributed to differences in diagnostic criteria, ACTH dosages (e.g., 250µg vs 1µg), patient populations (e.g., critically ill vs. stable outpatients), and geographical factors. The use of a low-dose ACTH test and inclusion of treatment-naïve patients in this study might contribute to detecting a higher prevalence of subclinical AI that might otherwise be missed.

A crucial finding of this study is the lack of a statistically significant difference in classical clinical features between HIV patients with AI and those with normal adrenal function. Symptoms like weight loss and weakness were common in both groups, suggesting that these are manifestations of HIV infection itself rather than specific indicators of AI in this population. This corroborates previous reports from Nigeria and India, which emphasize the need for a high index of suspicion and biochemical confirmation for AI diagnosis in HIV patients, as typical AI symptoms are often absent or indistinguishable from HIV progression.[9,25]

Conclusion

This study concludes that adrenocortical insufficiency is a common endocrinopathy among newly diagnosed, treatment-naïve HIV patients in Maiduguri, affecting approximately one-third of this population (33.3% based on ACTH stimulation). Critically, the classical clinical features of adrenal insufficiency often overlap with general symptoms of HIV infection, making clinical diagnosis unreliable. Therefore, a high index of suspicion and biochemical testing, particularly with a low-dose ACTH stimulation test using appropriate local reference values, are essential for accurate and early diagnosis of AI in HIV patients. This diagnostic vigilance is crucial for timely intervention to reduce associated morbidity and mortality.

The implementation of systematic AI screening programs, coupled with appropriate hormone replacement therapy when indicated, represents an essential component of comprehensive HIV care. Healthcare providers should maintain heightened clinical suspicion for AI in HIV patients presenting with compatible symptoms, even in the absence of advanced immunosuppression.

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