ORIGINAL ARTICLE

Clinical and Laboratory Features of Adrenocortical Insufficiency in HIV Patients: Insights from a Nigerian Cohort

Hassan AA,[1,2] Mubi BM,[1,2] Adamu AA,[3] Dungus MM,[4] Amali AO,[1,5] Musa AH[6]

ABSTRACT

Background: Adrenal dysfunction is common in human immunodeficiency virus (HIV) infection, but diagnosis is difficult due to the overlap of symptoms with advanced HIV disease. This study assessed the clinical and laboratory features of adrenal insufficiency (AI) in newly diagnosed, treatment-naïve HIV patients in Maiduguri, Nigeria. **Methods:** This cross-sectional study enrolled 138 stable HIV-infected adults and 132 apparently healthy adults as controls. Adrenal function was evaluated using the 1-µg low-dose short synacthen test, with serum cortisol measured at baseline and at 30 and 60 minutes. Fasting plasma glucose, full blood count, erythrocyte sedimentation rate (ESR) and serum electrolytes were also measured. Using a locally derived reference cut-off from the apparently healthy controls in this study, AI was defined as a peak cortisol of <504 nmol/L at 30 or 60 minutes and a cortisol increment of <124 nmol/L. **Results:** Weight loss (57.4%) and weakness (47.3%) were the most frequent symptoms among the HIV patients, with no significant difference between patients with AI and those with normal adrenal function. Compared with healthy controls, HIV patients had significantly lower fasting plasma glucose, serum sodium, bicarbonate, haemoglobin, body weight, and body mass index, while serum chloride and erythrocyte sedimentation rate were higher. However, no significant differences in anthropometric or laboratory parameters were observed between HIV patients with and without AI. **Conclusion:** The lack of distinction in classical clinical features and common laboratory parameters between HIV patients with and without AI underscores the difficulty in clinical differentiation. These findings emphasized the critical need for biochemical confirmation of AI in HIV patients using diagnostic tests like the low-dose short synacthen test.

Key words: Adrenal insufficiency, Human immunodeficiency virus, ACTH stimulation test, Weight loss, Weakness, Nigeria,

[1]Department of Medicine, College of Medical Sciences, University of Maiduguri. [2]Endocrinology, Diabetes and Metabolism Unit, Department of Internal Medicine, University of Maiduguri Teaching Hospital, Borno, Nigeria. [3]Infectious Disease Unit, Department of Internal Medicine, University of Maiduguri Teaching Hospital, Borno, Nigeria. [4]Department of Chemical Pathology, University of Maiduguri Teaching Hospital, Borno, Nigeria. [5]Pulmonology Unit, Department of Internal Medicine, University of Maiduguri Teaching Hospital, Borno, Nigeria. [6]Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, University of Maiduguri.

Corresponding Author:

AbdulFattah Alhaji Hassan
Phone: +2348032387865, Address: Department of Medicine, College of Medical Sciences, University of Maiduguri, abdulfattahhassan@unimaid.edu.ng

Received: 18th February 2026
Accepted: 6th May 2026
Published online: 30th June 2026

Access this article online

QuickResponse Code

website:www.bornomedicaljournal.com

DOI: 10.31173/bomj.bomj_2604_23

Introduction

Human immunodeficiency virus (HIV) infection is widely recognized for its diverse effects on various components of the endocrine system.[1,2] Adrenal dysfunction, specifically involving the hypothalamic-pituitary-adrenal (HPA) axis, is noted as the most common endocrinopathy in HIV/AIDS, with the adrenal gland itself being the most frequently involved site.[3,4] The underlying causes of HPA axis dysfunction in HIV/AIDS are complex, ranging from the direct effects of HIV infection, infiltration of glands by opportunistic infections or malignancies, to the side effects of highly active antiretroviral therapy (HAART) or other adjunct medications needed to treat or prevent opportunistic infections.[1,2]

Several studies have evaluated adrenal function in HIV-infected individuals, demonstrating a relatively high prevalence of both subclinical and overt adrenal insufficiency. In a Nigerian study conducted in Lagos, Odeniyi and colleagues assessed

adrenocortical function using a low-dose ACTH stimulation test and reported that approximately 34.8% of treatment-naïve HIV patients had biochemical evidence of adrenal insufficiency, despite many lacking overt clinical features.[4] Similarly, a larger study from Northern Nigeria by Akase *et al.* evaluated both the clinical and biochemical profile of adrenal insufficiency among HIV-infected patients and found that hypocortisolism was common, with prevalence rates of 30.9% using basal cortisol and 16.3% following ACTH stimulation. Notably, the study demonstrated poor correlation between clinical features and biochemical findings, highlighting the diagnostic challenges of AI in this population.[5] Beyond Nigeria, studies across Africa have consistently reported that adrenal insufficiency in HIV patients is frequently underdiagnosed due to nonspecific clinical manifestations that overlap with chronic HIV infection and opportunistic diseases.[6] These manifestations include fatigue, weight loss, anorexia, and electrolyte abnormalities, which may be misattributed to the underlying infection rather than adrenal dysfunction.[6]

A significant challenge in diagnosing AI in HIV-infected patients lies in the non-specific nature of its symptoms, which often overlap with the general manifestations of advanced HIV disease.[1,3,4,7–9] Consequently, clinical AI is infrequently diagnosed, often only after significant gland destruction has occurred (80-90%).[1,7,8] Therefore, a high index of suspicion and biochemical confirmation are paramount for timely diagnosis and intervention.[3]

Despite these important contributions, there remains a paucity of comprehensive studies in Nigeria that simultaneously evaluate both the clinical and laboratory characteristics of adrenocortical insufficiency, particularly in specific regional cohorts and treatment-naïve populations. Existing studies have either focused predominantly on biochemical assessment or have demonstrated inconsistencies between clinical presentation and laboratory findings.

This study focuses on the clinical and laboratory characteristics of adrenal insufficiency within a cohort of newly diagnosed, treatment-naïve HIV patients in Maiduguri, Nigeria. By examining these features, this study aims to provide insights into the presentation of AI in this specific population, highlighting the importance of biochemical evaluation for accurate diagnosis.

METHODS

This was a cross-sectional study involving newly diagnosed, treatment-naïve HIV patients conducted at the University of Maiduguri Teaching Hospital (UMTH) in Borno State, Nigeria.

Study Population and Eligibility Criteria: The target population included newly diagnosed treatment-naïve HIV-positive adults aged ≥18 years. Participants were excluded if they had been on any form of antiretroviral therapy, were on steroids, were acutely ill requiring admission, were pregnant, had concomitant diabetes mellitus, or declined consent. Healthy HIV-negative adults aged ≥18 years were also recruited as controls to establish normal reference values for serum cortisol.

Sample Size Determination: The sample size was calculated using Cochran's formula for proportions ($n = Z^2pq/d^2$), with a confidence level of 95% ($Z = 1.96$), an estimated prevalence of adrenal dysfunction among HIV patients of 10% ($p = 0.10, q = 0.90$), and a precision level of 5% ($d = 0.05$). Substituting these values yielded a minimum sample size of 138 participants. After adjusting for a 10% attrition rate, the final sample size was rounded to 153. Accordingly, 153 HIV-positive participants were recruited, along with an equal number of apparently healthy HIV-negative controls.[10] A total of 138 stable HIV patients and 132 healthy subjects completed the study, with data from 129 HIV-positive participants and 91 healthy subjects analyzed after outlier exclusion.

Data Collection: Data collection involved detailed history and physical examination, including biodata, presence of symptoms like weakness, fatigue, cough, fever, weight loss, anorexia, nausea, vomiting, diarrhoea, hyperpigmentation, and history of glucocorticoid or antiretroviral therapy. Physical measurements such as weight, height, and blood pressure (supine and erect) were taken.[11] HIV cases were staged according to the WHO clinical staging for HIV/AIDS.[12]

One microgram ACTH preparation

One microgram ACTH was obtained by dilution method. 1ml (250µg) ACTH (synacthen) was diluted with 249ml of 0.9% normal saline to produce 1µg/ml of ACTH. The storage of the diluted ACTH (synacthen) was at -4^{o}C according to the manufacturer's specifications.[4]

Screening and Diagnostic Criteria for Adrenal Insufficiency: Adrenal function was screened using a 1µg low-dose short synacthen test.[4] A 21-G cannula was inserted into a cubital vein and kept patent with heparinized saline. The subjects were then allowed to rest for 30 minutes after securing venous access before the sample collection. Low dose short synacthen test was performed as follows. A baseline blood samples for cortisol, fasting plasma glucose (FPG), full blood count (FBC), ESR and electrolytes were collected immediately before administration of ACTH (synacthen).[4] ACTH testing was conducted between 8:00 hours and 9:00 hours. After the samples have been taken, subjects received an intravenous injection of 1µg ACTH (synacthen). After the bolus ACTH administration, blood samples were drawn for cortisol level at 30 minutes and 60 minutes. The samples were centrifuged and separated and transported on an ice slab to the laboratory where the samples were stored at -20^{0}C until assayed. Serum cortisol levels were measured using an Enzyme-Linked Immunosorbent Assay (ELISA) (Calbiotech Inc. CA, USA, *Cat#: CO103S*).[13] Other laboratory tests included fasting plasma glucose (FPG) (glucose oxidase method),[14] serum electrolytes (sodium, potassium, chloride, bicarbonate) (automated ion selective electrode method),[15] serum urea (enzymatic method),[16,17] , creatinine by colorimetric method [18](modified Jaffe method), and full blood count (FBC) and erythrocyte sedimentation rate (ESR) were also collected.

The normal reference values for serum cortisol were derived from the 91 healthy subjects who had peak serum cortisol ≥ 500 nmol/L at 30 or 60 minutes post 1µg ACTH stimulation.

Normal response to the 1µg short synacthen test was defined as peak cortisol post-ACTH stimulation of ≥ 504 nmol/L plus an increment from basal to a stimulated cortisol level of ≥124 nmol/L.

Adrenal insufficiency (AI) was defined as basal serum cortisol of < 207.56 nmol/L, or peak serum cortisol 30 or 60 minutes 1µg post ACTH stimulation of < 504 nmol/L, plus a post-stimulation increment of < 124 nmol/L.

Ethical approval was obtained from the ethics and research committee of the University of Maiduguri Teaching Hospital (approval number UMTH/REC/387). All participants provided informed consent after receiving detailed information about the study. Confidentiality was maintained by using participant IDs instead of names, and the study adhered to the Helsinki Declaration.[19]

Data Analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 23.0. Summary descriptive statistics were presented as mean ± standard deviation for normally distributed continuous data and median (interquartile range) for skewed continuous data. Continuous data were compared using a t-test, while categorical data were compared using a chi-square test. Statistical significance was set at p-value < 0.05.

RESULTS

Sociodemographic Characteristics of Study Participants

HIV-positive participants were significantly older than healthy controls. Gender distribution differed, with more females in the HIV group. Marital status varied, with married and single individuals predominating among controls, while widowed and divorced were more frequent among HIV patients. Educational attainment also differed: secondary and tertiary education were more common among controls, whereas primary and no formal education were higher among HIV participants as shown in Table 1. Age and sex matching was not achieved due to recruitment challenges in the immediate post–COVID-19 period.

Table 1: Sociodemographic Characteristics of Study Participants

Variable	HIV Positive N=138	Healthy Subjects N=132	P-value
Gender*	n (%)	n (%)	**0.040**
Male	58 (42.0)	72 (54.5)	
Female	80 (58.0)	60 (45.5)	
Age (Mean±SD)	36.07±8.87	31.91±11.18	**0.001**
Marital Status			**<0.001**
Single	36 (26.1)	62 (47.0)	
Married	52 (37.7)	68 (51.5)	
Divorced	20 (14.5)	0 (0.0)	
Widowed	30 (21.7)	2 (1.5)	
Level of Education			**<0.001**
Primary	14 (10.1)	4 (3.0)	
Secondary	55 (39.9)	60 (45.5)	
Tertiary	39 (28.3)	63 (47.7)	
No Formal Education	30 (21.7)	5 (3.8)	

χ^2 =4.234, df = 1, p = 0.040;
HIV, human immunodeficiency virus; SD, standard deviation.

Baseline Frequency of Adrenocortical Insufficiency: Using the derived cut-off values, the study found that 43 (33.3%) of the 129 newly diagnosed treatment-naïve HIV patients had adrenal insufficiency after ACTH stimulation, while 86 (66.7%) had a normal response. When using basal cortisol levels, 68 (52.7%) were classified as having AI, as shown in Table 2.

Table 2: Prevalence of Adrenal Insufficiency at 0- and 30- 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)

AI, adrenal insufficiency.

Clinical Features of Adrenocortical Insufficiency among HIV Patients: The most common clinical features among all HIV patients in the cohort were weight loss (57.4%) and weakness (47.3%), as shown in Table 3. Less common features included nausea (5.4%) and postural hypotension (4.7%).

Importantly, there was no statistically significant difference in the distribution of clinical features between HIV patients with AI and those with normal adrenal function. For instance, weakness was present in 51.2% of AI patients versus 45.3% of non-AI patients, and hyperpigmentation in 14.3% of AI patients versus 9.3% of non-AI patients, but these differences were not statistically significant.

Table 3: 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.

Baseline Laboratory Characteristics:

As shown in Table 4, HIV-positive patients had significantly lower body weight and BMI compared to healthy controls (both $p < 0.001$). The mean body weight of HIV-positive individuals was 8.65 kg lower than that of controls (95% CI: −11.77 to −5.50; $p < 0.001$), while BMI was reduced by a mean of 2.74 kg/m² (95% CI: −3.92 to −1.55; $p < 0.001$).

Several laboratory parameters also differed significantly between the two groups. Fasting plasma glucose (FPG), serum sodium, bicarbonate (HCO_3^-), haemoglobin, and eosinophil percentage were significantly lower among HIV-positive patients, whereas serum chloride and erythrocyte sedimentation rate (ESR) were significantly higher. Serum potassium and creatinine levels did not differ significantly between the two groups.

The fasting plasma glucose (FPG) was significantly lower in HIV-positive patients, with a mean difference of −0.58 mmol/L compared with controls (95% CI: −0.75 to −0.42; $p < 0.001$). Serum sodium levels were modestly but significantly lower in the HIV-positive patient with a mean difference of −3.04 mmol/L (95% CI: −5.77 to −0.30; $p = 0.030$). In contrast, serum chloride levels were significantly higher among HIV-positive patients, with a mean difference of +1.51 mmol/L (95% CI: 0.49 to 2.55; $p = 0.004$), while bicarbonate (HCO_3^-) levels were slightly but significantly lower with a mean difference of −0.44 mmol/L (95% CI: −0.83 to −0.04; $p = 0.029$).

Table 4: Baseline Anthropometry and Laboratory Characteristics of HIV Patients and Healthy Subjects

Variables	HIV Positive Mean±SD / IQR	Healthy Controls Mean±SD / IQR	Md/ U test	95% CI	P-value
Anthropometry					
Weight (Kg)	58.03±11.63	66.68±11.47	-8.650	-11.77 to -5.5	**<0.001**
BMI (Kg/m²)	20.97±4.14	23.71±4.69	-2.735	-3.92 to -1.55	**<0.001**
Biochemical					
FPG (mmol/L)	4.48±0.53	5.07±0.73	- 0.583	-0.75 to -0.42	**<0.001**
Na (mmol/L)	138.90±12.59	141.93±4.80	- 3.035	- 5.77 to – 0.30	**0.030**
K (mmol/L)	4.22±0.64	4.30±0.57	- 0.080	- 0.24 to 0.09	0.341
Chloride (mmol/L)	103.95±4.04	102.44±3.70	1.514	0.49 to 2.55	**0.004**
HCO_3 (mmol/L)	21.34±1.51	21.78±1.40	-0.439	-0.83 to -0.04	**0.029**
Creatinine (µmol/L) *	82.0 (65.0-111.0)	83.0 (65.0 – 94.0)	5335.0		0.250
Urea (mmol/L) *	4.1 (3.4 – 5.4)	3.5 (3.0 – 4.6)	4315.0		**0.001**
Haematological					
Hb (g/dL)	10.98±2.09	13.20±1.60	-2.22	-2.734 to - 1.71	**<0.001**
ESR (mm/HR) *	80.0 (41.0 – 117.0)	6.0 (2.0 – 18.0)	840.5		**<0.001**
Eosinophils (%)	1.0 (1.0 – 2.0)	2.0 (1.0 – 2.0)	4004.5		**<0.001**

**, median (interquartile range).*

CI, confidence interval; Cr, creatinine; ESR, erythrocyte sedimentation rate; FPG, fasting plasma glucose; Hb, haemoglobin; HCO_3, bicarbonate; HIV, human immunodeficiency virus; K, potassium; M±SD, mean plus or minus standard deviation; Md, mean difference; Na, sodium.

Median urea concentration was significantly higher in HIV-positive patients (p = 0.001). Haemoglobin levels were markedly reduced in the HIV-positive group, with a mean difference of −2.22 g/dL compared with controls (95% CI: −2.73 to −1.71; p < 0.001). Markers of systemic inflammation were substantially elevated among HIV-positive patients, as evidenced by a significantly higher median ESR (p < 0.001). Eosinophil percentage also differed significantly between the two groups (p < 0.001), been lower in HIV-positive patients.

Table 5: Baseline Anthropometry and Laboratory Characteristics of HIV Patients with and without Adrenocortical Insufficiency

Variables	AI Mean±SD / IQR	No AI Mean±SD / IQR	Md / U Test	95% CI	P-value
Anthropometry					
Weight (Kg)	60.7 ± 12.9	56.7 ± 10.8	3.93	-0.33 – 8.19	0.070
BMI (Kg/m²)	21.8 ± 4.4	20.5 ± 3.9	1.29	-0.23 – 2.81	0.096
Biochemical					
FPG (mmol/L)	4.54 ± 0.50	4.46 ± 0.55	0.084	-0.11 – 0.28	0.400
Na (mmol/L)	137.23 ± 21.59	139.73 ± 2.48	- 2.50	-0.92 – 4.16	0.453
K (mmol/L)	4.23 ± 0.57	4.21 ± 0.67	0.023	-0.21 – 0.26	0.846
Chloride (mmol/L)	104.09 ± 4.24	103.88 ± 3.95	0.21	-1.29 – 1.71	0.782
HCO_3 (mmol/L)	21.12 ± 1.77	21.41 ± 1.37	- 0.198	-0.81 – 0.42	0.522
Creatinine (µmol/L)*	83.0 (63.0 – 108.0)	81.0 (65.0 – 113.0)	1815.5		0.867
Urea (mmol/L)*	4.1 (3.6 – 6.1)	4.1 (3.3 – 4.9)	1663.5		0.354
Haematological					
Hb (g/dL)	11.33 ± 1.86	10.80 ± 2.18	0.52	-0.25 – 1.29	0.18
ESR (mm/HR)*	80.0 (15.0 – 103.0)	83.0 (50.0 – 120.0)	1468.5		0.057
Eosinophils (%)*	1.0 (1.0 – 2.0)	1.0 (1.0 – 2.0)	1799.5		0.786

**, median (interquartile range).*

AI, adrenal insufficiency; CI, confidence interval; Cr, creatinine; ESR, erythrocyte sedimentation rate; FPG, fasting plasma glucose; Hb, haemoglobin; HCO_3, bicarbonate; HIV, human immunodeficiency virus; K, potassium; M±SD, mean plus or minus standard deviation; Md, mean difference; Na, sodium.

As presented in Table 5, the study found no significant differences in anthropometric (weight, BMI), biochemical (FPG, Na+, K+, Chloride, HCO3, Creatinine, Urea), or haematological (Hb, ESR, Eosinophils) parameters between HIV patients with AI and those with normal adrenal functions. This suggests that these common laboratory abnormalities in HIV patients are not specific indicators of co-existing AI.

Discussion

The findings of this study provide crucial insights into the presentation of adrenal insufficiency in a Nigerian cohort of newly diagnosed, treatment-naïve HIV patients. The high prevalence of AI (33.3% after ACTH stimulation) at baseline among newly diagnosed HIV patients aligns with previous research in Nigeria, such as Odeniyi et al., who reported a 34.8% prevalence in Lagos.[4] This underscores that AI is a significant, yet potentially overlooked, comorbidity in this population.

A critical observation from this study is the lack of statistically significant differences in classical clinical features between HIV patients with and without biochemically confirmed AI. Symptoms such as weight loss and weakness, while predominant in the overall HIV cohort (57.4% and 47.3% respectively), were equally common in both groups, as were nausea, postural hypotension, and hyperpigmentation. This aligns with prior Nigerian studies by Odeniyi et al.[4] and Akase et al.[5], which also noted that most HIV patients with AI did not present with classic symptoms. Globally, Sharma et al. [20] emphasized that AI diagnosis requires a high index of suspicion because typical symptoms are often absent or overlap with other conditions.[21] The clinical presentation of AI, characterized by fatigue, weight loss, nausea, and

postural hypotension, is highly consistent with symptoms of advanced HIV infection.[1,9] This overlap makes clinical differentiation extremely challenging, emphasizing the need for biochemical evaluation for a definitive diagnosis.[3]

Regarding laboratory features, the study found significant differences in several parameters when comparing HIV patients to healthy subjects, with HIV patients exhibiting lower weight, BMI, FPG, serum Na+, HCO3, Hb, and eosinophils, and higher serum chloride and ESR. These findings corroborate previous research by Odeniyi et al.[4] which also noted anthropometric and biochemical differences between HIV patients and healthy controls.

However, the lack of significant differences in these anthropometric and laboratory parameters between HIV patients with AI and those with normal adrenal function is a crucial finding. This indicates that routine blood tests, often used to monitor general health in HIV patients, while reflecting the overall impact of HIV, are not reliable indicators for distinguishing those with AI from those with normal adrenal function. This observation is consistent with studies by Akase et al.[5] and Ekpebegh et al.[22] in Africa, and Madi et al.[23] more broadly, which similarly found no distinct differences in these parameters between HIV patients with and without AI. This further reinforces that the presence of AI in HIV patients cannot be reliably determined by clinical signs or common laboratory tests alone.

Therefore, to ensure accurate and timely diagnosis of AI in HIV patients, a high index of suspicion and confirmation through appropriate laboratory evaluation, specifically using the ACTH stimulation test, remains indispensable.[3]

Conclusion

This study reveals that adrenocortical insufficiency is common in newly diagnosed treatment-naïve HIV patients in Maiduguri, Nigeria, affecting approximately one-third of this cohort. The classical clinical features suggestive of AI, such as weight loss, weakness, nausea, and hyperpigmentation, are frequently observed in HIV patients irrespective of their adrenal status, making clinical differentiation challenging. Furthermore, common anthropometric and laboratory parameters, while differing significantly between HIV patients and healthy individuals, do not significantly differ between HIV patients with and without AI, rendering them poor indicators for diagnosing AI. These findings collectively emphasize the critical need for a high index of suspicion for AI in all HIV patients and underscore the imperative for biochemical confirmation using diagnostic tests like the low-dose short synacthen test for early diagnosis and appropriate intervention.

Limitations

1. The study population was strictly defined as newly diagnosed, treatment-naïve HIV patients. As participants on any form of antiretroviral therapy were excluded, and because the side effects of highly active antiretroviral therapy (HAART) or other medications are known causes of hypothalamic-pituitary-adrenal (HPA) axis dysfunction, these findings may not be generalizable to HIV patients already receiving treatment. Also, the geographical scope was limited to a health centre in Maiduguri, Borno State, Nigeria, which restricts the generalizability of the results to other global HIV populations.
2. Opportunistic infections and malignancies were not screened, and imaging was not performed to identify different aetiologies of adrenocortical dysfunction.
3. No matching for age and sex

References

1. Bhatia E. Adrenal disorders in people with HIV: The highs and lows. Indian Journal of Medical Research. 2018 Jan 1;147(2):125–7. doi:10.4103/IJMR.IJMR_1087_17 PubMed PMID: 29806599.
2. Manosuthi W, Ongwandee S, Bhakeecheep S, Leechawengwongs M, Ruxrungtham K, Phanuphak P, et al. Guidelines for antiretroviral therapy in HIV-1 infected adults and adolescents 2014, Thailand. AIDS Res Ther. 2015 Apr 24;12(1):1–9. doi:10.1186/s12981-015-0053-z PubMed PMID: 25908935.
3. Prasanthai V, Sunthornyothin S, Phowthongkum P, Suankratay C. Prevalence of adrenal insufficiency in critically ill patients with AIDS. J Med Assoc Thai. 2007;90(9):1768–74. PubMed PMID: 17957917.
4. Odeniyi IA, Fasanmade OA, Ajala MO, Ohwovoriole AE. Adrenocortical function in Nigerians with human immunodeficiency virus infection. Ghana Med J. 2013;47(4):171–7. PubMed PMID: 24669022.
5. Akase IE, Habib AG, Bakari AG, Hamza M, Gezawa ID. The prevalence and clinical profile of adrenocortical deficiency among HIV infected

persons in Northern Nigeria. Afr Health Sci. 2019;19(2):1947–52. doi:10.4314/ahs.v19i2.18 PubMed PMID: 31656478.

6. Nassoro DD, Mkhoi ML, Sabi I, Meremo AJ, Lawala PS, Mwakyula IH. Adrenal Insufficiency: A Forgotten Diagnosis in HIV/AIDS Patients in Developing Countries. Int J Endocrinol. 2019;2019(2342857):1–9. doi:10.1155/2019/2342857
7. Mayo J, Collazos J, Martínez E, Ibarra S. Adrenal Function in the Human Immunodeficiency Virus–Infected Patient. Arch Intern Med. 2002;162(10):1095. doi:10.1001/archinte.162.10.1095 PubMed PMID: 12020177.
8. Zapanti E, Terzidis K, Chrousos G. Dysfunction of the hypothalamic-pituitary-adrenal axis in HIV infection and disease. Hormones. 2008;7(3):205–16. doi:10.14310/horm.2002.1200 PubMed PMID: 18694859.
9. Afreen B, Khan KA, Riaz A. Adrenal Insufficiency In Pakistani Hiv Infected Patients. J Ayub Med Coll Abbottabad. 2017;29(3):428–31. PubMed PMID: 29076676.
10. Cochran 1977 Sampling Techniques Third Edition | PDF [Internet]. [cited 2026 Apr 11]. Available from: https://www.scribd.com/document/534046574/Cochran-1977-Sampling-Techniques-Third-Edition
11. World Health Organization. WHO steps surveillance manual: the WHO stepwise approach to chronic disease risk factor surveillance. WHO; 2005.
12. Centre for Disease Control and Prevention. WHO Clinical Staging of HIV / AIDS. Cdc. 2015;12–4.
13. Cortisol ELISA | Calbiotech [Internet]. [cited 2022 Jun 30]. Available from: https://calbiotech.com/collections/diagnostic-assays/products/cortisol-elisa
14. Mosby Co. St. Louis. Baltimore. Philadelphia. Toronto. Glucose (Mono Reagent) (GOD / POD method) Red Quinone dye + H 2 O. Vols. 854–856. 1989;854–856:8–9.
15. Garcia RA, Vanelli CP, Pereira Junior O dos S, Corrêa JO do A. Comparative analysis for strength serum sodium and potassium in three different methods: Flame photometry, ion-selective electrode (ISE) and colorimetric enzymatic. J Clin Lab Anal. 2018 Nov 1;32(9). doi:10.1002/jcla.22594 PubMed PMID: 29921038.
16. Urea | Reagent | Randox Laboratories [Internet]. [cited 2022 Jun 30]. Available from: https://www.randox.com/urea/
17. Fawcett, J.K. and Scott, J.E. JClinP. Urease-Berthelot Method Colorimetric Method. Anal Chem. 1960;2(13):2–5.
18. Chromý V, Rozkošná K, Sedlák P. Determination of serum creatinine by Jaffe method and how to calibrate to eliminate matrix interference problems. Clin Chem Lab Med. 2008 Aug 1;46(8):1127–33. doi:10.1515/CCLM.2008.224 PubMed PMID: 18724810.
19. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013 Nov 27;310(20):2191–4. doi:10.1001/JAMA.2013.281053 PubMed PMID: 24141714.
20. Sharma N, Sharma LK, Anand A, Gadpayle AK, Gaurav K, Mukherjee S, et al. Presence, patterns & predictors of hypocortisolism in patients with HIV infection in India. Indian J Med Res. 2018 Feb 1;147(2):142. doi:10.4103/IJMR.IJMR_43_16 PubMed PMID: 29806602.
21. Shashidhar P, Shashikala G. Low dose adrenocorticotropic hormone test and adrenal insufficiency in critically ill acquired immunodeficiency syndrome patients. Indian J Endocrinol Metab. 2012;16(3):389. doi:10.4103/2230-8210.95680
22. Ekpebegh CO, Ogbera AO, Longo-Mbenza B, Blanco-Blanco E, Awotedu A, Oluboyo P. Basal cortisol levels and correlates of hypoadrenalism in patients with human immunodeficiency virus infection. Medical Principles and Practice. 2011 Oct;20(6):525–9. doi:10.1159/000330022 PubMed PMID: 21986010.
23. Madi DR, Khanapure S, Ramapuram J, Achappa B, Rao S. Adrenal insufficiency in patients with acquired immunodeficiency syndrome-an underestimated problem. Retrovirology. 2012;9(Suppl 1):P143. doi:10.1186/1742-4690-9-S1-P143

Cite this Article as: Hassan AA, Mubi BM, Adamu AA, Dungus MM , Amali AO, Musa AH. Clinical and Laboratory Features of Adrenocortical Insufficiency in HIV Patients: Insights from a Nigerian Cohort. **Bo Med** J 2026; 23 (1): 71-78 **Source of Support:** Nil, **Conflict of Interest:** None declared