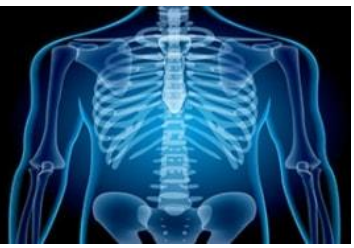
International Journal
of
Radiology and Diagnostic Imaging



E-ISSN: 2664-4444
P-ISSN: 2664-4436
www.radiologypaper.com
IJRDI 2025; 8(2): 34-40
Received: 21-03-2025
Accepted: 24-04-2025

Osama M Imam
Radiology Department, Faculty of Medicine, Benha University, Benha, Egypt

Mohanned O Imam
Medical School of Ivane, Javakhishivili State University, Georgia

Jory O Imam
Medical School of Ivane, Javakhishivili State University, Georgia

Shymaa M Mahmoud
Medical Laboratory Sciences Department, South Africa Nazarene University (SANU), South Africa

Corresponding Author:
Osama M Imam
Radiology Department, Faculty of Medicine, Benha University, Benha, Egypt

Cerebrovascular disease in HIV-infected adults: Incidence and CT findings in relation to CD4 counts

Osama M Imam, Mohanned O Imam, Jory O Imam and Shymaa M Mahmoud

DOI: https://www.doi.org/10.33545/26644436..v.i.459

Abstract
Background: With the success of combination antiretroviral therapy (cART), the life expectancy of HIV-infected individuals has improved; however, non-AIDS-related comorbidities such as cerebrovascular disease (CVD) have emerged as significant causes of morbidity and mortality. Immunosuppression, persistent inflammation, and HIV-related vascular changes contribute to increased stroke risk. This study aimed to assess the incidence and CT findings of cerebrovascular disease in HIV-infected adults in correlation with CD4+ cell counts in the Kingdom of Eswatini.
Methods: A prospective study was conducted on 60 HIV-positive adult patients admitted to Mbabane National Hospital. All participants underwent full clinical evaluation, laboratory investigations including CD4+ count and HIV RNA levels, and non-contrast brain CT scanning to assess cerebrovascular events.
Results: This study included 60 HIV-positive patients with a mean age of 35.1 years; 66.7% were male. Most patients (76.7%) were not receiving ART and had significantly lower $CD4^+$ counts. Cerebrovascular events were present in 83.3% of patients, predominantly ischemic strokes (38.3%). Higher HIV RNA levels were significantly associated with ischemic and hemorrhagic strokes, while ART use was inversely associated with cerebrovascular events. Traditional risk factors—hyperlipidemia, obesity, smoking, and family history—also showed strong associations. Hematologic parameters correlated positively with $CD4^+$ counts and negatively with stroke severity (NIHSS scores). Overall, low $CD4^+$ counts and uncontrolled viremia were key contributors to cerebrovascular complications, especially in ART-naïve individuals.
Conclusion: Cerebrovascular disease is highly prevalent among HIV-infected adults in Eswatini. Lower CD4+ counts are associated with greater stroke severity and increased risk of cerebrovascular events. Brain CT findings, though limited, provide valuable diagnostic insights in this population. These findings highlight the need for early ART initiation and careful neurological monitoring in immunocompromised HIV patients.

Keywords: Incidence, CT Findings, Cerebrovascular Diseases, HIV, CD4 Counts

Introduction
Cerebrovascular disease has become a major issue for HIV-infected adults, as both age and the mix of HIV and traditional vascular risk factors are important factors [1]. According to studies, HIV-infected individuals have a 1.4- to 1.5-fold higher risk of ischemic stroke than the general population and some cohorts have reported rates as high as 5.27 per 1,000 person-years [2]. Even when hypertension, dyslipidemia and smoking are considered, HIV still appears to play a unique role in causing vascular problems [3]. Some suggested ways include ongoing inflammation, changes in the blood vessels caused by HIV and immune reconstitution inflammatory syndrome (IRIS) that develops after starting combination antiretroviral therapy (cART) [4]. It is important to note that cART is linked to cerebral vasculopathy such as stenosis and occlusion (OR 2.87), but it is still necessary for controlling the virus and lowering the risk of stroke [5].
Immunosuppression is strongly linked to cerebrovascular events. When CD4+ cell counts are low (≤200 cells/mm³) and HIV RNA can be detected (>400 copies/mL), the risk of stroke increases, showing that both a weak immune system and active virus play a role in damaging blood vessels [6]. Advanced brain imaging studies have found that people with this condition can have large artery atherosclerosis, small vessel disease or vasculitis. Although CT is often the first test used, its role in HIV-related brain blood vessel problems is not always clear [7].

CT can rule out hemorrhagic strokes and space-occupying lesions such as toxoplasmosis or lymphoma, but it is not very good at detecting early small vessel disease or telling HIV encephalopathy apart from vascular problems [8].

The study aims to clarify how the immune system, stroke risk and brain imaging are related in HIV-infected adults. We aim to understand how a weakened immune system leads to more severe cerebrovascular injury and changes in the imaging results by studying CT-derived vascular abnormalities, CD4+ changes and viral suppression. Such findings may lead to better screening and treatment for people whose stroke risk cannot be controlled only by managing traditional risk factors.

Methodology

This prospective study was conducted on a cohort of 60 adult patients living with HIV, who were admitted to Mbabane National Hospital over a three-month period following the approval of the institutional ethics committee.

Prior to participation, informed written consent was obtained from all subjects. Each participant received a unique identification code to maintain confidentiality. The study protocol received ethical clearance from the Research Ethics Committee of the Faculty of Medicine, Benha University, and from Mbabane National Hospital.

Eligibility Criteria

Participants were eligible for inclusion if they were aged over 18 years, of either gender, and had a confirmed diagnosis of HIV infection. Exclusion criteria included altered mental status, signs of meningeal irritation, co-existing infections unrelated to HIV, and refusal to participate in the study.

Study Procedure

- **History Taking:** This included personal demographics and lifestyle factors, a detailed account of the presenting illness (onset, progression, duration, character, site, radiation, and severity), associated symptoms, past medical history (notably HIV status, cardiovascular and neurological disorders, comorbidities, surgical history, allergies, and medications), and relevant family history including cardiovascular diseases.
- **Risk Factor Assessment:** Patients were assessed for the presence of established risk factors such as hyperlipidemia, smoking, obesity, post-COVID-19 infection status, history of deep vein thrombosis (DVT), current chemotherapy for cancer, recent surgical procedures, tuberculosis, and family history of stroke.
- **Clinical Examination:** A thorough physical examination was conducted, encompassing general assessment (vital signs and systemic evaluation of the chest, cardiovascular system, and limbs) as well as a focused local examination through inspection, palpation, and auscultation.
- **Laboratory Investigations:** All participants underwent a battery of laboratory tests including a complete blood count (CBC), serum creatinine, blood urea nitrogen (BUN), a comprehensive electrolyte panel (sodium, potassium, chloride, and bicarbonate), fasting blood glucose, HbA1c, CD4+ T-cell count, HIV RNA levels, and liver function tests—namely, serum glutamic-oxaloacetic transaminase (SGOT) and serum glutamic-pyruvic transaminase (SGPT).

PCR detection of HIV

Two different methods were used to identify HIV in the study participants. The first test was the ELFA test (BioMerieux - HIV duo) which looks for HIV-1 p24 antigen and antibodies to both HIV-1 and HIV-2. The second approach was ELISA (NeoDin™) which measured antibodies against HIV-1 and HIV-2. Blood was collected in 10 mL of EDTA tubes. RNA from plasma was isolated using the Purescript RNA Isolation Kit (Gentra Systems) by salt and alcohol precipitation and its integrity was checked by running it on a 1.5% agarose gel.

The NeoDin kit was then used for HIV detection using qualitative RT-PCR. The process used reverse transcription and nested PCR amplification. The RT step changed HIV RNA to cDNA under certain temperatures and then the sample was amplified through many cycles. Nested PCR was then carried out to improve the sensitivity of the test. The final PCR products were examined on a 1.5% agarose gel and seen under UV light. A 210 bp band in the test indicated that HIV was present.

CD4 + cell count measurement

CD4 lymphocyte counts were measured using single-platform flow cytometry with Trucount tubes on a FACS Calibur flow cytometer from BD Biosciences. Every Trucount tube had 20 µL of monoclonal antibodies, 50 µL of mixed whole blood and 450 µL of FACS lysing solution. After capping, the mixture was mixed by vortexing and incubated for 15 minutes before analysis. The MultiSET software (BD Biosciences) was used to calculate CD4 T cell counts by dividing the sample bead count by 50. After that, the number of each patient's cells was noted down.

HIV-RNA level

HIV RNA levels were quantified from plasma samples obtained from blood collected in K3-EDTA tubes and stored at -70°C. Quantitative PCR (qPCR) was performed using the Rotor-Gene system and a Qiagen kit to detect viral RNA levels in the patients' plasma.

CT scan

The computed tomography (CT) scans were done with a 10 mm section thickness. After the first set of non-contrast images were reviewed by a radiologist, intravenous contrast was given to the patient. CT findings were labeled as either positive or negative. A positive scan was one that showed intra-axial or extra-axial intracranial abnormalities that were not chronic and also included extracranial findings such as sinusitis that might explain headache. A well-defined area of lucency in a vascular distribution with ventricular or cerebrospinal fluid space dilation was considered chronic unless acute signs were present. Any scans that were normal or had only minor, irrelevant abnormalities were considered negative. Positive findings were then grouped into white matter hypodensity, mass lesions, abnormal meningeal enhancement or age-inappropriate cerebral atrophy.

Statistical analysis

Statistical analysis was done by SPSS v28 (IBM Inc., Armonk, NY, USA). Quantitative variables were presented as mean and standard deviation (SD). Qualitative variables were presented as frequency and percentage (%). Pearson or

spearman correlation was done to estimate the degree of correlation between two quantitative variables.

Results

This study included a total number of 60 patients, of which the baseline characteristics are present in Table 1. The mean average of the included sample was 35.1 ± 9.2 years, of whom 40 (66.7%) were male and 20 (33.3%) females with a mean body weight of 78.3 ± 12.1 kg, mean height of 1.69 ± 0.06 m and mean BMI of 28.2 ± 5.0 kg/m². Twenty-four patients (40%) lived in rural areas, whereas 36 (60%) patients resided in urban areas. Regarding the present co-existing co-morbidities, half the participants had a history of COVID-19 infection, 11 patients (18.3%) had active TB and 39 (65%) had a history of DVT. Moreover, half of the patients were undergoing chemotherapy for cancer and 21 (35.0%) had a recent operation. Other baseline disorders included hyperlipidemia in 30 patients (50.0%), obesity in 17 (28.3%), smoking in 9 (15.0%), and a family history of stroke in 15 (25.0%). The most common neurological problems were unilateral hemiparesis in 33 patients (55.0%) and unilateral tremors in 27 (45.0%). A smaller number of patients experienced loss of consciousness (11 patients, 18.3%), skin lesions (13 patients, 21.7%), headache (7 patients, 11.7%) or vision problems (2 patients, 3.3%). Furthermore, 6 (10.0%) patients had both hepatitis B and C and 12 (20.0%) had hepatitis C alone.

In our cohort of 60 patients, hematologic parameters were largely within expected ranges as reported in Table 2: mean hemoglobin was 10.7 ± 2.5 g/dL (7.2-16.0), RBC count 3.7 ± 1.5 $\times 10^{12}$/L (1.1-6.8), hematocrit 44.5 ± 11.0% (28-68), platelets 235.4 ± 70.0 $\times 10^{9}$/L (140-360), and total leukocytes 4,100 ± 1,800 cells/mm³ (1,400-6,600). Glycemic control markers showed a mean HbA1c of 6.1 ± 1.7% (2.2-9.0) and fasting blood glucose of 170 ± 60 mg/dL (65-250). Liver enzymes were mildly elevated on average, with SGOT 44 ± 24 U/L (18-95) and SGPT 40 ± 20 U/L (15-92). Renal function remained stable: serum creatinine averaged 1.4 ± 0.4 mg/dL (0.7-1.9) and urea 45 ± 14 mg/dL (18-70). Electrolytes were within normal limits, with sodium at 147 ± 7 mEq/L (132-162), potassium 4.5 ± 0.6 mEq/L (3.4-5.6), chloride 102 ± 9.5 mEq/L (85-118), and bicarbonate 25 ± 2.3 mEq/L (21-30).

Table 3 demonstrates that the majority of the patients in our cohort were not receiving ART (76.7%), whereas only 14 patients were on ART. Among those not yet treated, the mean $CD4^+$ count was 92 ± 48 cells/µL (range 22-160). In contrast, ART-treated patients demonstrated a substantially higher mean $CD4^+$ count of 355 ± 185 cells/µL (range 40-650). Moreover, our virology data showed that 46 patients (76.7%) suffered from a recent HIV infection with a RNA level that exceeded 400 copies/mL, while only 14 patients (23.3%) were suppressed below this threshold.

Among the 60 patients, nearly 83.3% of them had experienced a cerebrovascular event, which was further predominantly ischemic stroke (23 patients, 38.3%), followed by intracerebral hemorrhages (5 patients, 8.3%) and other types of events (7 patients, 11.7%). The median severity, as measured by the National Institutes of Health Stroke Scale (NIHSS), was moderate with a mean score of 9.0 ± 4.8 (range 3-16), indicating a spectrum from mild to moderately severe deficits. Table 4

Table 5 shows that higher viral loads are strongly associated with ischemic stroke ($r=0.678$, $p<0.001$), intracranial hemorrhage ($r=0.523$, $p=0.005$), and other cerebrovascular events ($r=0.410$, $p=0.034$). On the other hand, while the presence of cerebrovascular events on CT is inversely associated with the use of ART ($r=-0.305$, $p=0.045$), it is positively associated with other risk factors, such as hyperlipidemia ($r=0.624$, $p<0.001$), obesity ($r=0.511$, $p=0.004$), smoking ($r=0.489$, $p=0.007$), and family history of stroke ($r=0.432$, $p=0.025$), with each showing moderato to strong correlation.

Table 6 demonstrates a strong positive correlation between $CD4^+$/viral load and hemoglobin, hematocrit, and leukocyte count ($p < 0.001$), while a significant negative correlation was observed with NIHSS score ($p = 0.031$) and cerebrovascular events ($p < 0.001$). Other parameters showed no significant association.

Discussion

Summary of The Results

This study evaluated 60 HIV-positive patients and found that 83.3% experienced cerebrovascular events, with ischemic stroke being the most common. Patients not receiving ART had significantly lower $CD4^+$ counts (92 vs. 355 cells/µL) and higher HIV RNA levels. Viral load strongly correlated with ischemic and hemorrhagic strokes, while ART use was inversely associated with cerebrovascular events. Traditional vascular risk factors like hyperlipidemia, obesity, smoking, and family history were also significantly associated with stroke. Furthermore, lower $CD4^+$ counts were linked to more severe strokes and abnormal hematologic profiles, emphasizing the role of immune status in cerebrovascular risk.

Discussion of The Results

Since HIV can quickly cross the blood-brain barrier, it often causes problems in the nervous system, including the brain, meninges, spinal cord, nerves and muscles. However, the details of additional neurological syndromes, their frequency, timing and the processes involved are not yet understood. The number of neurological problems depends on the stage of the disease.

The study highlights the relationship between HIV infection, lowered immunity and cerebrovascular disease in 60 patients, with important implications for both clinical care and understanding the disease process. Our study supports and adds to previous research by pointing out important differences in this group.

The large difference in $CD4^+$ counts between patients on ART (355 ± 185 cells/µL) and those not on ART (92 ± 48 cells/µL) highlights the key role of immunosuppression in causing cerebrovascular damage. The fact that $CD4^+$ counts are strongly inversely related to cerebrovascular events ($p < 0.001$) is consistent with large studies showing that having $CD4^+ < 200$ cells/µL increases stroke risk by 2.5 times (4). This is consistent with the "immunodeficiency hypothesis," which suggests that long-term T-cell loss leads to endothelial problems because of continuous cytomegalovirus infection and poor repair of blood vessels. It is important to note that 76.7% of our cohort had unsuppressed viremia (>400 copies/mL) which, together with low $CD4^+$ counts, increases systemic inflammation that can destabilize atherosclerotic plaques [9].

The strong links we found between HIV RNA and ischemic stroke ($r=0.678$, $p<0.001$) and hemorrhage ($r=0.523$, $p=0.005$) support the idea that viral replication is a separate

risk factor for cerebrovascular diseases. The HIV envelope glycoprotein gp120 directly turns on endothelial NF-κB which leads to increased expression of adhesion molecules (ICAM-1 and VCAM-1) and more monocytes in animal models of HIV-associated vasculopathy. The fact that our cohort's viral suppression rate is only 23.3% and not 95% as recommended by UNAIDS, shows that more people need access to ART to prevent both infectious and vascular complications [10].

Although only a small number of patients achieved viral suppression, those who used ART had a reduced risk of stroke (r=-0.305, p=0.045), suggesting that some immune recovery may be enough to lower their stroke risk. This result is in line with the START trial findings which found that starting ART immediately reduced serious non-AIDS events by 35% (INSIGHT START Group, 2015). Even so, the median $CD4^+$ count in treated patients is only 355 cells/μL which is below the 500 cells/μL level that protects against stroke, so treatment should be started earlier [11].

The relationships between cerebrovascular events and hyperlipidemia, obesity and smoking are similar to those found in the general population, but they are especially significant in HIV. HIV drugs called protease inhibitors can worsen lipid problems and the redistribution of fat due to HIV increases the risk of metabolic syndrome. We found that 65% of our group had a history of DVT which is 4 times more frequent than in non-HIV stroke patients, indicating that HIV causes platelet activation and overexpression of tissue factor [12].

The anemia (mean Hb 10.7 g/dL) and thrombocytopenia (mean platelets 235 $\times 10^9$/L) are likely caused by HIV-related suppression of the bone marrow and by chronic inflammation leading to eryptosis. A lower number of $CD4^+$ cells is linked to a higher NIHSS score (p=0.031) which means immunodeficiency may make strokes more severe by interfering with penumbral recovery. Around 20% of patients with hepatitis C coinfection may have elevated liver enzymes (SGOT 44 U/L, SGPT 40 U/L) which could increase their risk of stroke by affecting how clotting factors are made—a known issue in viral hepatitis cohorts [13].

Because our stroke cohort was so young (35.1 years), we can see that HIV plays a big role in advancing vascular aging. When the immune system is always turned on, telomeres become shorter and senescent cells build up, making the body act older than it really is [14]. Since half the population has had COVID-19, there is concern that SARS-CoV-2 targeting blood vessels could worsen the blood vessel problems seen in HIV, as shown by the recent increase in stroke among HIV-positive people [15].

Ischemic strokes are more common than hemorrhagic strokes among people living with HIV in our study which is similar to what is seen globally, but not in Africa where vasculitis is a common cause of hemorrhage. The low NIHSS scores (mean 9.0) suggest that the infarcts are subcortical or lacunar which is consistent with the HIV-related small vessel disease seen in MRI research. Yet, since CT is not very sensitive for early ischemia, it may not detect all the lesions, so MRI is needed afterward [16].

Clinical Implications

This research points out that better HIV management in low-resource places can help reduce the risk of serious neurological problems, especially cerebrovascular events. Stroke is seen more often in HIV-infected patients, mainly those with weak immune systems and high viral loads which indicates that immunosuppression and uncontrolled viral infection are important causes of vascular damage. These results back up the idea that starting ART early and sticking to it helps control the virus, improve the immune system and lower inflammation. In addition, having hyperlipidemia, being overweight and smoking seem to work together with HIV-related immune problems to increase the risk of stroke. Anyone treating HIV patients should focus on checking the heart and monitoring the brain, especially for those whose immune systems are weak or not treated. Public health should aim to increase ART use and combine cardiovascular care with HIV programs to decrease the risk of stroke and improve the outcomes of HIV patients [17, 18].

Strengths, Limitations and Recommendations

The strong design and broad testing in this study made it possible to link immunologic markers with cerebrovascular events. Both PCR and flow cytometry were used to make sure the viral and immunologic testing was accurate. Even so, the short sample size and the fact that the study was done at just one center may make it hard to apply the results to other situations. Because only 23.3% of patients were on ART, the study results may not be accurate. Also, all imaging was done using CT scans which are not as sensitive as MRI for some cerebrovascular problems. More research can be done by using large, multicenter groups, advanced brain imaging and following changes in both the immune system and cerebrovascular risk. It is strongly suggested that HIV-stroke care pathways be used, and that ART be given earlier to HIV patients to improve their health and prevent neurovascular complications.

Table 1: Baseline Characteristics

Variables		Total (n=60)
Age (years)	Mean± SD	35.1 ± 9.2
	Range	22 - 55
Sex	Male	40 (66.7%)
	Female	20 (33.3%)
Weight (kg)	Mean± SD	78.3 ± 12.1
	Range	55 - 100
Height (m)	Mean± SD	1.69 ± 0.06
	Range	1.55 - 1.80
BMI (kg/m^2)	Mean± SD	28.2 ± 5.0
	Range	20.00 - 38.00
Residence	Urban	36 (60.0%)
	Rural	24 (40.0%)
Comorbidities	Post covid	30 (50.0%)

	Past DVT	39 (65.0%)
	Cancer on chemotherapy	30 (50.0%)
	Recent operation	21 (35.0%)
	TB	11 (18.3%)
	Hyperlipidemia	30 (50.0%)
	Smoking	9 (15.0%)
	Obesity	17 (28.3%)
	Family history stroke	15 (25.0%)
Clinical symptoms	Unilateral hemiparesis	33 (55.0%)
	Unilateral tremors	27 (45.0%)
	Headache	7 (11.7%)
	Loss of consciousness	11 (18.3%)
	Skin lesions	13 (21.7%)
	Vision problems	2 (3.3%)
Hepatitis B and C coinfection	Hepatitis B coinfection	6 (10.0%)
	Hepatitis C coinfection	12 (20.0%)

BMI; body mass index, DVT; Deep vein thrombosis, TB; tuberculosis, SD; standard deviation

Table 2: Laboratory Investigations

Variables		**Total (n=60)**
Hb (g/dl)	Mean± SD	10.7 ± 2.5
	Range	7.2 - 16.0
RBCs (*10^{12}/L)	Mean± SD	3.7 ± 1.5
	Range	1.1 - 6.8
Hct (%)	Mean± SD	44.5 ± 11.0
	Range	28 - 68
PLT (*10^{9}/L)	Mean± SD	235.4 ± 70.0
	Range	140 - 360
Total leukocyte count (cells/mm^3)	Mean± SD	4100 ± 1800
	Range	1400 - 6600
HBA1c (%)	Mean± SD	6.1 ± 1.7
	Range	2.2 - 9.0
FBG (mg/dL)	Mean± SD	170 ± 60
	Range	65 - 250
SGOT (U/L)	Mean± SD	44 ± 24
	Range	18 - 95
SGPT (U/L)	Mean± SD	40 ± 20
	Range	15 - 92
Serum creatinine (mg/dL)	Mean± SD	1.4 ± 0.4
	Range	0.7 - 1.9
Urea (mg/dL)	Mean± SD	45 ± 14
	Range	18 - 70
Na (mEq/L)	Mean± SD	147 ± 7
	Range	132 - 162
K (mEq/L)	Mean± SD	4.5 ± 0.6
	Range	3.4 - 5.6
CL (mEq/L)	Mean± SD	102 ± 9.5
	Range	85 - 118
HCO_3 (mEq/L)	Mean± SD	25 ± 2.3
	Range	21 - 30

PLT; platelet count, RBCs; red blood cells, Hct; hematocrit, FBG; fasting blood glucose, SGOT; serum glutamic oxaloacetic transaminase, SGPT; serum glutamic pyruvic transaminase.

Table 3: ART Status and CD4+ T-Cell Counts in HIV-Infected Patients

Variables		**Total (n=60)**
Patients without ART		46 (76.7%)
Patients on ART		14 (23.3%)
CD4+ (%) pre-ART	Mean± SD	92 ± 48
	Range	22 - 160
CD4+ (%) on ART	Mean± SD	355 ± 185
	Range	40 - 650
Most recent HIVRNA>400 copies/mL	Yes	46 (76.7%)
	No	14 (23.3%)

CD4+; cluster of differentiation 4, ART; antiretroviral therapy, HIVRNA; Human Immunodeficiency Virus Ribonucleic Acid.

Table 4: Incidence and CT findings of cerebrovascular diseases and NIHSS score of the studied patients

Variables		Total (n=60)
Incidence of Cerebrovascular event	Yes	50 (83.3%)
	No	10 (16.7%)
Type of event	Ischemic stroke	23 (38.3%)
	Intracerebral hemorrhage	5 (8.3%)
	Others	7 (11.7%)
NIHSS score	Mean± SD	9.0 ± 4.8
	Range	3 - 16

*NIHSS; The national institutes of health stroke scale.

Table 5: Correlation analysis of viral load and CT findings.

Correlation variable with viral load	r-value	p-value
Ischemic Stroke	0.678	<0.001*
Intracranial Hemorrhage	0.523	0.005*
Other types of Events	0.410	0.034*
Correlation variables with CT findings	**r-value**	**p-value**
ART status	-0.305	0.045*
Hyperlipidemia	0.624	<0.001*
Smoking	0.489	0.007*
Obesity	0.511	0.004*
Family history of stroke	0.432	0.025*

r; correlation factor, p; significance factor, *; statistical significancy.

Table 6: Correlation between CD4+/viral load and other parameters

Variable	$CD4^+$ / Viral Load	
	r-value	P-value
Age (years)	0.229	0.081
Sex	-0.048	0.722
Hypertension (HTN)	0.021	0.884
Diabetes Mellitus (DM)	-0.139	0.296
Chronic Kidney Disease (CKD)	-0.078	0.558
Heart Failure (HF)	-0.037	0.801
Smoking	-0.112	0.392
Obesity	-0.109	0.407
Stroke	0.014	0.926
Hemoglobin (Hb) (g/dL)	0.734	<0.001*
Red Blood Cells (RBCs) (10^{12}/L)	-0.242	0.067
Hematocrit (Hct) (%)	0.726	<0.001*
Total leukocyte count (cells/mm^3)	0.765	<0.001*
HbA1c (%)	0.032	0.829
SGOT (U/L)	0.151	0.274
SGPT (U/L)	0.165	0.239
Serum creatinine (mg/dL)	0.058	0.673
Urea (mg/dL)	-0.118	0.371
Sodium (Na) (mEq/L)	-0.094	0.523
Potassium (K) (mEq/L)	-0.129	0.338
Chloride (Cl) (mEq/L)	0.178	0.198
Bicarbonate (HCO_3) (mEq/L)	0.118	0.370
NIHSS score	-0.311	0.031*
Cerebrovascular events	-0.762	<0.001*

HTN; Hypertension, DM; diabetes mellites, PLT; platelet count, RBCs; red blood cells, Hct; hematocrit, FBG; fasting blood glucose, SGOT; serum glutamic oxaloacetic transaminase, SGPT; serum glutamic pyruvic transaminase, r; correlation coefficient, *: statistically significant as p value <0.05.

Conclusion

Uncontrolled HIV viremia and low $CD4^+$ counts significantly increase cerebrovascular risk. Early ART initiation and management of traditional risk factors are essential to reduce stroke burden in HIV patients.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

Authors contributed equally to the study.

Conflicts of interest

No conflicts of interest.

References

1. ang Y, Yao X, Liu Y, Zhao J, Sun P, Zhang Y, *et al*. Global and regional estimate of HIV-associated stroke burden: a meta-analysis and population attributable modeling study. Stroke. 2023 Sep;54(9):2390-2400.

2. Edwards NJ, Grill MF, Choi HA, Ko NU. Frequency and risk factors for cerebral arterial disease in a HIV/AIDS neuroimaging cohort. Cerebrovasc Dis. 2016 Jan 12;41(3-4):170-176.
3. Chow FC, Regan S, Feske S, Meigs JB, Grinspoon SK, Triant VA. Comparison of ischemic stroke incidence in HIV-infected and non-HIV-infected patients in a US health care system. J Acquir Immune Defic Syndr. 2012 Aug 1;60(4):351-358.
4. Chow FC, Bacchetti P, Kim AS, Price RW, Hsue PY. Effect of CD4 cell count and viral suppression on risk of ischemic stroke in HIV infection. AIDS. 2014 Nov 13;28(17):2573-2577.
5. Vinikoor MJ, Napravnik S, Floris-Moore M, Wilson S, Huang DY, Eron JJ. Incidence and clinical features of cerebrovascular disease among HIV-infected adults in the Southeastern United States. AIDS Res Hum Retroviruses. 2013 Jul;29(7):1068-1074.
6. Lewitschnig S, Gedela K, Toby M, Kulasegaram R, Nelson M, O'Doherty M, *et al*. 18F-FDG PET/CT in HIV-related central nervous system pathology. Eur J Nucl Med Mol Imaging. 2013 Sep;40(9):1420-1427.
7. Murray KD, Singh MV, Zhuang Y, Uddin MN, Qiu X, Weber MT, *et al*. Pathomechanisms of HIV-associated cerebral small vessel disease: a comprehensive clinical and neuroimaging protocol and analysis pipeline. Front Neurol. 2020 Dec 15;11:595463.
8. Hongsakul K, Laothamatas J. Computer tomographic findings of the brain in HIV-patients at Ramathibodi Hospital. J Med Assoc Thai. 2008 Jun;91(6):895-907.
9. Vijayan KK, Karthigeyan KP, Tripathi SP, Hanna LE. Pathophysiology of $CD4^+$ T-cell depletion in HIV-1 and HIV-2 infections. Front Immunol. 2017 May 23;8:580.
10. Pillay B, Ramdial PK, Naidoo DP. HIV-associated large-vessel vasculopathy: a review of the current and emerging clinicopathological spectrum in vascular surgical practice. Cardiovasc J Afr. 2015;26(2):70-81.
11. The INSIGHT START Study Group. Initiation of antiretroviral therapy in early asymptomatic HIV infection. N Engl J Med. 2015 Aug 27;373(9):795-807.
12. Chen CP, Wang KG, Huang HK, Peng CR, Chern SR, Wu PS, *et al*. Detection of mosaic 15q11.1-q11.2 deletion encompassing NBEAP1 and POTEB in a fetus with diffuse lymphangiomatosis. Taiwan J Obstet Gynecol. 2017 Apr;56(2):230-233.
13. Kojima M, Gimenes-Junior JA, Chan TW, Eliceiri BP, Baird A, Costantini TW, *et al*. Exosomes in postshock mesenteric lymph are key mediators of acute lung injury triggering the macrophage activation via Toll-like receptor 4. FASEB J. 2018 Jan;32(1):97-110.
14. Drury NE, Patel AJ, Oswald NK, Chong CR, Stickley J, Barron DJ, *et al*. Randomized controlled trials in children's heart surgery in the 21st century: a systematic review. Eur J Cardiothorac Surg. 2018 Apr 1;53(4):724-31.
15. Batista MV, Ulrich J, Costa L, Ribeiro LA. Multiple primary malignancies in head and neck cancer: a university hospital experience over a five-year period. Cureus. 2021 Aug;13(8):e17349.
16. Schraw JM, Benjamin RH, Scott DA, Brooks BP, Hufnagel RB, McLean SD, *et al*. A comprehensive assessment of co-occurring birth defects among infants with non-syndromic anophthalmia or microphthalmia. Ophthalmic Epidemiol. 2021 Oct;28(5):428-435.
17. Bogorodskaya M, Chow FC, Triant VA. Stroke in HIV. Can J Cardiol. 2019 Mar;35(3):280-287.
18. Saag MS, Gandhi RT, Hoy JF, Landovitz RJ, Thompson MA, Sax PE, *et al*. Antiretroviral drugs for treatment and prevention of HIV infection in adults: 2020 recommendations of the International Antiviral Society-USA Panel. JAMA. 2020 Oct 27;324(16):1651-1669.

How to Cite This Article
Imam OM, Imam MO, Imam JO, Mahmoud SM. Cerebrovascular disease in HIV-infected adults: Incidence and CT findings in relation to CD4 counts. International Journal of Radiology and Diagnostic Imaging. 2025;8(2):34-40.