The Egyptian Journal of Hospital Medicine (April 2025) Vol. 99, Page 2442-2447

# Stroke in the HIV Population: Immunologic Mechanisms, Clinical Patterns, and Neuroimaging Approaches: Review Article

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## ABSTRACT

**Background:** With the advent of combination antiretroviral therapy (cART), the survival of people living with HIV (PLWH) has improved substantially. However, cerebrovascular disease has emerged as a major non-AIDS-related comorbidity. Mounting evidence implicates immune suppression, inflammation, and vascular dysfunction in the elevated stroke risk observed among this population.

**Objective:** To explore the relationship between HIV infection, particularly CD4 count levels, and the risk, presentation, and radiological diagnosis of cerebrovascular disease in PLWH.

**Methods:** A comprehensive literature review was conducted through PubMed, Scopus, and Web of Science databases up to May 2025. Search terms included "HIV," "stroke," "CD4 count," "cerebrovascular disease," and "CT imaging." Eligible studies included peer-reviewed original research, systematic reviews, and relevant clinical guidelines. Emphasis was placed on the diagnostic utility of neuroimaging and the impact of immunologic status on cerebrovascular outcomes.

**Conclusion:** HIV-related cerebrovascular disease is multifactorial, with low CD4 counts serving as both a risk marker and a predictor of atypical stroke presentations. Neuroimaging, particularly non-contrast and contrast-enhanced computed tomography (CT), is critical for timely diagnosis and treatment planning. Integrating immunologic markers with imaging findings may improve risk stratification and outcomes in PLWH. Further research is needed to refine preventive and therapeutic approaches tailored to this population.

**Keywords:** HIV, Stroke, CD4 Count, Neuroimaging, Cerebrovascular Disease.

## INTRODUCTION

The use of antiretroviral therapy (ART) has led to longer life spans for people with HIV and fewer deaths from opportunistic infections and AIDS-related cancers. As HIV-positive individuals grow older, diseases such as cardiovascular disease (CVD) are now major causes of sickness and death [1]. Adults with HIV are more likely to have a heart attack and tend to experience it at a younger age than those without HIV [2]. Some factors are HIV-related vascular damage from long-term inflammation and the activity of T cells and macrophages in blood vessels and the high rate of traditional CVD risk factors (such as smoking, high blood pressure, and dyslipidaemia) in high-risk individuals [3].

Cerebrovascular events (CVEs), which include ischemic strokes, haemorrhagic strokes, and transient ischemic attacks, have received less attention in people living with HIV. Before the use of ART, infections in the central nervous system made it difficult to see how HIV alone raised the risk of stroke. Because ART was reducing infections, it was expected that CVEs would decrease [4]. However, from 1997 to 2006, the number of people with HIV who had strokes went up. Because stroke subtypes are all different, evaluating cerebrovascular risk factors in HIV is difficult, so it is important to confirm each outcome. It is important to study how CD4 cell counts relate to the outcomes of confirmed ischemic stroke [5].

HIV-positive patients often undergo urgent cranial CT scans to rule out acute pathology [6]. While several studies have explored CT timing and appropriateness in selected groups, few have identified clinical variables—such as CD4 count—that predict, which HIV-infected patients will benefit most from head CT. To address this gap, investigators assessed the diagnostic yield of screening head CTs among HIV-positive individuals stratified by CD4 count [7].

### Gross Brain Anatomy

The brain, protected by the skull and cushioned in cerebrospinal fluid, is the central organ of the nervous system. Alongside the spinal cord, it makes up the CNS, which controls body activities by processing sensory information and sending out instructions. At birth, the brain weighs about 350–400 grams, approximately 25% of its adult weight, reaching about 1.4–1.45 kg by age 10–15. The brain undergoes rapid growth in the first three years, reaching nearly 90% of adult size by age five, although it continues to change subtly throughout life, influenced by both internal and external factors [8].

**The brain has three main parts: the cerebrum, cerebellum, and brainstem:**

1) The cerebrum is the biggest part and it has many grooves (sulci) and ridges (gyri) on its surface to increase its area. The brain is composed of two hemispheres, which are joined by the corpus callosum and it is divided into four lobes. At the front of the brain is the frontal lobe, which is responsible for reasoning, controlling movements, thinking deeply, and speaking verbally. The parietal lobe, which is in the middle,



Received: 12/01/2025
Accepted: 12/03/2025

handles sensations such as touch, pressure, and pain. The auditory cortex and hippocampus, which support hearing and memory, are found in the temporal lobe at the bottom of the brain. The occipital lobe, which is at the back, manages visual processing [9].

2) The cerebellum, located beneath the cerebrum, behind the brainstem, and divided into two hemispheres, controls balance, posture, and coordinated movement. It receives input from sensory systems and fine-tunes motor activity [10].

3) The brainstem, the oldest part evolutionarily, connects the brain and spinal cord. It includes the medulla oblongata, pons, and midbrain. Just above it lies the diencephalon, which contains structures like the thalamus (a relay for sensory signals), the hypothalamus (regulating endocrine and autonomic functions), the epithalamus (including the pineal gland for melatonin production), and the subthalamus (linked to motor control) [11].

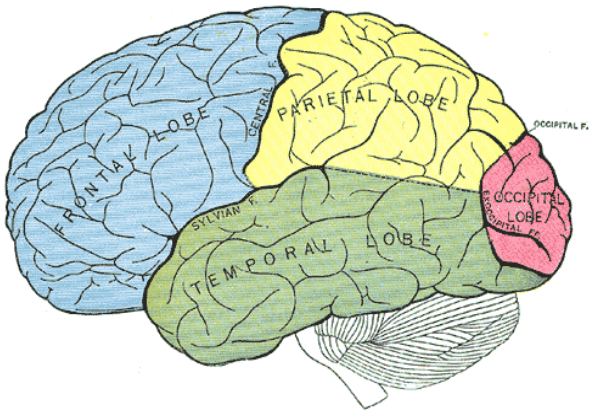


**Figure 1:** Lobes of cerebrum [12].

The limbic system, primarily located on the medial side of the temporal lobe, includes important structures such as the amygdala and hippocampus, which are central to processing emotions, learning, and memory. Another key component at the base of the brain is the pituitary gland, a small but powerful endocrine organ that regulates many essential body functions through hormone secretion. Additionally, the brain connects to the body via twelve pairs of cranial nerves, which mainly manage motor and sensory functions in the head and neck, although some extend their influence to other regions [1].

The brain's blood supply is maintained by the internal carotid and vertebral arteries, which come together at the base of the brain to form the Circle of Willis — a critical collateral circulation system that helps ensure stable blood flow. Major arteries branching from this system, including the anterior, middle, and posterior cerebral arteries, provide blood to different brain regions, and any disruption can result in stroke, leading to motor or sensory impairments. Venous drainage occurs through a specialized network of dural venous sinuses, which ultimately empty into the internal jugular veins. Unlike most veins in the body, these sinuses lack valves, and they collect blood from the brain's superficial and deep veins, ensuring efficient removal of deoxygenated blood [10].

**Cerebrovascular Disease**

A stroke, or cerebrovascular accident (CVA), occurs when blood flow to part of the brain is abruptly disrupted, leading to tissue injury. Approximately 85% of strokes are ischemic, resulting from vessel occlusion, while the remainder are haemorrhagic. Stroke incidence and related mortality have declined over recent decades, yet it remains the leading cause of adult disability worldwide. Rapid recognition is critical because interventions such as intravenous thrombolysis (within 4.5 hours) and mechanical thrombectomy (within 6 hours, extendable to 24 hours in select large-vessel occlusions) can markedly improve outcomes. Every moment of delay allows more brain tissue to be lost, reinforcing the adage, "time is brain" [2].

Ischemic strokes are categorized as embolic, thrombotic, or lacunar. Major modifiable risk factors include hypertension (the most common), diabetes, smoking, obesity, elevated LDL cholesterol, and atrial fibrillation. Chronic uncontrolled hypertension, particularly prevalent and often earlier in African-American populations, causes small-vessel strokes in regions such as the internal capsule and pons [3]. Lifestyle measures—weight loss, salt restriction, and a diet rich in fruits and vegetables—can lower blood pressure, with each 10 mm Hg reduction correlating to a one-third reduction in primary stroke risk. Elevated LDL predisposes to intracerebral plaque formation and thrombotic infarctions. In older adults, atrial fibrillation increasingly accounts for cardioembolic strokes. Haemorrhagic strokes, which make up about 15%, arise from causes such as uncontrolled hypertension, aneurysm rupture, arteriovenous malformations, illicit drug use, and amyloid angiopathy [13].

Upon presentation, determining the exact time of symptom onset or last-seen-normal is paramount for treatment eligibility. A coordinated rapid assessment—including the National Institutes of Health Stroke Scale (NIHSS)—focuses on consciousness, language, motor strength (e.g., pronator drift), visual fields, gaze, facial movement, and cerebellar function. Initial imaging involves a non-contrast head CT to exclude haemorrhage, followed by CT angiography and perfusion studies to delineate infarct core versus penumbra. Early CT signs of ischemia, such as a hyperdense middle cerebral artery segment, guide urgent decisions [14]. If no haemorrhage is

detected, IV tPA is administered promptly. Subsequent evaluation for mechanical thrombectomy leverages perfusion imaging, especially in large-vessel occlusions. Beyond acute management, all patients receive antiplatelet therapy, statins, and risk factor optimization. Further workup—including ECG, echocardiography, lab panels, telemetry, and vascular imaging—aims to identify the specific stroke aetiology and guide secondary prevention [15].

**HIV Infection and its Impact on Cerebrovascular Health**

In the past decade, middle- to low-income countries have seen a 100% rise in stroke cases, thanks to HIV as well as the usual vascular risk factors and an older population. HIV can affect the risk and causes of stroke and using combination antiretroviral therapy (cART) may directly speed up atherosclerosis and indirectly boost stroke risk by increasing life expectancy. About 1–5% of people with HIV have a stroke during their lifetime, but 4–34% of them have brain lesions found during autopsy [16]. There has been a 43% increase in the number of stroke patients with HIV infection admitted to hospitals in the USA in the past nine years. Those with HIV who have strokes tend to be much younger than those without HIV, with a median age of 42.9 years in some US studies, which suggests different causes than those normally seen in strokes. Regular exposure to HIV with low levels of the virus and ongoing low-level inflammation may increase the risk of stroke, mainly in areas where HIV is common [17].

Stroke in HIV-positive patients is usually described in the same way as in HIV-negative people—sudden and localized symptoms—but they also tend to show atypical signs such as confusion, fever, loss of consciousness, and gradual worsening of symptoms over time. There are more ischemic than haemorrhagic strokes in hospital series from sub-Saharan Africa and ischemic stroke accounts for over 90% of strokes in HIV patients [18]. Before cART was used, the number of cerebral hemorrhages and ischemic infarctions was similar, though this may be because of people using drugs or the way patients were admitted. HIV may lead to stroke by making blood clot more easily and by causing heart-related blood clots from HIV-related heart disease, endocarditis or narrowing of the arteries [19].

Patients with HIV who are immunosuppressed are more likely to develop stroke from infections such as Mycobacterium tuberculosis, varicella zoster virus and syphilis which may not always show skin symptoms. HIV-associated vasculopathy involves problems with both intracranial and extracranial arteries such as fusiform aneurysms, narrowing, blood clots, thickening of the inner lining, and damage to the elastic lamina which are often found in young HIV patients without atherosclerosis [20].

HIV leads to problems in the endothelium which results in both vascular disease and atherogenesis. Continuous activation of the endothelium by HIV-1 proteins, infected CD4+ T cells, monocytes, macrophages and viral-induced proinflammatory cytokines leads to increased vessel wall permeability, more leukocytes entering the walls, continuous inflammation and changes in the inner lining of blood vessels. Chemokine ligand 2 (CCL2) brings leukocytes to the brain and HIV-positive patients have higher plasma levels of VCAM1, ICAM1, E-selectin, hsCRP, interleukin 6 and cystatins, showing that HIV-1 may be involved in activating endothelial cells [21].

A disorder in von Willebrand factor, thrombomodulin, plasminogen activator inhibitor-1 antigen, tissue factor, and d-dimer encourage blood clotting which is seen more in patients who have high anti-p24 antibody levels and severe disease. They can speed up atherosclerosis or lead to vascular disease directly which is why HIV infection is linked to problems in the brain's blood vessels [22].

**CD4 Counts in HIV-Related Morbidity and Mortality**

CD4 counts are a cornerstone in evaluating immune status in HIV-infected individuals, reflecting the number of CD4-positive T-helper lymphocytes per cubic millimeter of blood. As HIV replicates, it targets and depletes CD4 cells, leading to progressive immunodeficiency. In the cART era, baseline CD4 count at therapy initiation informs clinical risk, but the latest absolute CD4 count more closely correlates with mortality. Effective ART suppresses viral replication, allowing CD4 counts to rebound and reduce the likelihood of opportunistic infections, which become more frequent as CD4 levels decline [23].

In the context of cerebrovascular health, lower CD4 counts are linked to heightened risk of both ischemic and haemorrhagic strokes. Chronic HIV-induced inflammation and immune activation drive endothelial dysfunction and atherogenesis. HIV-1 proteins (e.g., TAT, GP120) and proinflammatory cytokines continuously activate the vascular endothelium, increasing permeability, leukocyte invasion, and intimal remodelling. Elevated markers such as VCAM1, ICAM1, E-selectin, hsCRP, and interleukin-6 in HIV-positive patients underscore this inflammatory milieu, which accelerates atherosclerosis and predisposes to vascular injury [24].

HIV infection also creates a prothrombotic state via derangements in coagulation factors—von Willebrand factor, thrombomodulin, plasminogen activator inhibitor-1, tissue factor, and d-dimer—correlating with disease severity. These coagulation abnormalities, alongside

traditional cardiovascular risk factors (smoking, hypertension, and dyslipidemia), further increase cerebrovascular disease risk. Additionally, HIV-associated coagulopathies (protein C/S deficiencies, and antiphospholipid antibodies) and cardioembolic sources (dilated cardiomyopathy, and endocarditis) contribute to stroke pathogenesis [25].

Furthermore, opportunistic infections such as Mycobacterium tuberculosis, varicella zoster virus, and syphilis, which occur more often in immunosuppressed HIV patients, can directly cause cerebrovascular events. Thus, fluctuations in CD4 counts not only mark HIV progression but also serve as prognostic indicators for cerebrovascular complications, highlighting the need for vigilant monitoring and risk factor management in this population [26].

**Diagnostic Imaging in Cerebrovascular Disease among HIV Patients**

HIV infection leads to progressive immunodeficiency by depleting CD4-positive T lymphocytes. Normal CD4 counts range from 500 to 1,500 cells/mm³, but persistently low levels (<200 cells/mm³) indicate advanced disease (AIDS) and high risk for opportunistic infections. In cerebrovascular imaging, CT revolutionized diagnosis by allowing non-invasive evaluation of brain parenchyma and vascular structures, although angiography remains essential for detailing vessel morphology and planning interventions [27].

Understanding CT attenuation values (expressed in Hounsfield units [HU]) is critical when interpreting scans in HIV-associated cerebrovascular disease. Brain parenchyma typically measures around 33 HU, with white matter at approximately 30 HU and gray matter at about 36 HU. Cerebrospinal fluid appears between 6 and 10 HU, while circulating blood measures roughly 54 HU owing to hemoglobin. Acute clotted blood can reach 60–100 HU; it gradually declines over 7–30 days as liquefaction occurs. Contrast agents elevate blood attenuation substantially (about +26 HU per 100 mg iodine/100 cm³), whereas normal brain tissue increases by only 3–4 HU due to the intact blood–brain barrier. At lower kilovoltage settings (e.g., <120 kVp), high-atomic-number materials like calcium appear relatively denser, aiding distinction between calcifications, and hemorrhage [28].

Conventional CT techniques pivot on non-contrast CT (NCCT) and contrast-enhanced CT (CECT). NCCT is first-line in acute cerebrovascular evaluation because blood is hyperdense relative to brain tissue, making hemorrhage detection rapid and reliable. It also visualizes calcifications and bony structures, crucial in assessing trauma or chronic vascular changes. The typical scan takes only minutes, enabling faster clinical decisions, such as ruling out hemorrhage prior to thrombolytic therapy in stroke patients [27].

CECT involves intravenous iodinated contrast, which highlights vascular anatomy and pathologies. It is particularly valuable for detecting aneurysms, arteriovenous malformations (AVMs), and stenoses. In ischemic stroke, CECT can identify vessel occlusion and infarct margins, guiding eligibility for interventions like mechanical thrombectomy. It also distinguishes tumor vascularity and surrounding edema, vital when evaluating HIV-related CNS neoplasms. Postoperative imaging of stents or coils likewise relies on CECT to assess device positioning and detect complications such as endoleaks [29].

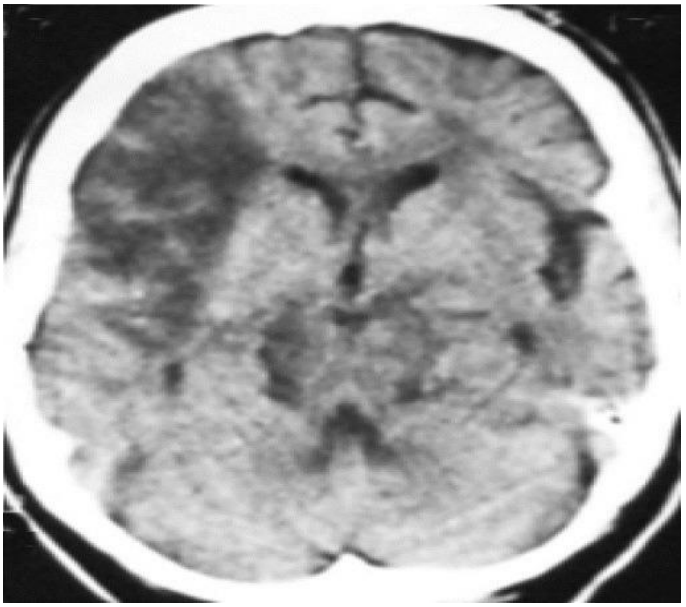

**Figure 2: CT scan image of the brain showing infarct-like lesion in the right frontal-parietal lobe in a patient with HIV [30].**

Computed tomography angiography (CTA) builds on CECT by specifically targeting vascular imaging through rapid, high-resolution acquisition during contrast passage. CTA accurately delineates arterial and venous structures, aiding in the diagnosis of stenosis, occlusion, aneurysms, and AVMs. Advanced techniques like four-dimensional CTA (4D-CTA) enhance temporal resolution, allowing dynamic assessment of flow patterns; this is beneficial in conditions such as Moya Moya disease, where collateral circulation evaluation guides staging and follow-up [31].

Perfusion CT (PCT) further extends CT capabilities by measuring cerebral blood flow (CBF), cerebral blood volume (CBV), and mean transit time (MTT). By capturing contrast dynamics, PCT identifies hypoperfused yet salvageable penumbral tissue in acute ischemia, informing reperfusion therapy decisions. Dual-energy CT (DECT) also shows promise in differentiating

active hemorrhage from contrast extravasation, since bleeding and iodine appear similarly hyperdense on routine scans. DECT's material decomposition maps can separate water-based from iodine-based signals, improving diagnostic confidence in hemorrhagic transformation and distinguishing calcified plaques from contrast [29,31].

Modern CT scanners typically perform helical acquisitions where detectors and X-ray tubes spin around the patient as the table advances. Reconstruction algorithms generate two-dimensional greyscale slices, with windowing adjustments (brain, bone, soft tissue) optimizing visualization of different structures. Slice thickness varies: thicker slices (e.g., 5 mm) enhance tissue contrast, while thinner slices (0.5–1.25 mm) improve spatial resolution. Bone algorithms apply edge enhancement to depict osseous details, important in trauma or suspected bone pathology [31].

### Future Prospectives

As the global population of people living with HIV (PLWH) continues to age, future research should prioritize longitudinal studies that integrate immunologic, vascular, and neuroimaging biomarkers to better predict cerebrovascular risk. Personalized risk stratification models incorporating CD4 count dynamics, inflammatory markers, and advanced imaging modalities—such as perfusion CT and MRI—may enhance early detection and intervention strategies. Additionally, prospective multicenter trials are needed to evaluate the efficacy of targeted preventive therapies, including antithrombotic regimens and aggressive cardiovascular risk factor modification, specifically tailored to the HIV population. Expanding research in low- and middle-income countries, where the burden of both HIV and stroke is high, will be essential for global applicability of findings and equitable care advancement.

## CONCLUSIONS

HIV-related cerebrovascular disease is multifactorial, with low CD4 counts serving as both a risk marker and a predictor of atypical stroke presentations. Neuroimaging, particularly non-contrast and contrast-enhanced computed tomography (CT), is critical for timely diagnosis and treatment planning. Integrating immunologic markers with imaging findings may improve risk stratification and outcomes in PLWH. Further research is needed to refine preventive and therapeutic approaches tailored to this population.

**Financial support and sponsorship: Nil.**
**Conflict of Interest: Nil**.

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