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Case Report of Cerebral Infarction with HIV

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HIV, Intracranial Hemorrhage, Stroke

ABSTRACT
Stroke is a serious neurological disorder caused by the obstruction or rupture of cerebral blood vessels, leading to impaired blood flow, brain tissue damage, and functional deficits. Globally, stroke is the second leading cause of death, affecting approximately 13.7 million people and resulting in 5.5 million deaths annually. Individuals with HIV infection are at increased risk of stroke due to mechanisms such as opportunistic infections, vasculopathy, cardioembolism, and coagulopathy, which exacerbate cerebrovascular vulnerability. This case report aims to explore the occurrence of stroke in a patient with a history of HIV infection and to examine the potential interplay between HIV-related factors and stroke pathophysiology. A detailed clinical evaluation, imaging studies, and review of the patient's medical history were conducted to document the stroke event, its presentation, and contributing risk factors. The findings demonstrate a clear association between the patient's HIV status and the stroke event, highlighting how HIV-related vascular and hematological changes may have contributed to the cerebrovascular insult. This case report underscores the importance of comprehensive management strategies for HIV-positive patients to monitor and mitigate stroke risk. It also emphasizes the need for increased awareness among clinicians regarding the potential for cerebrovascular complications in this population. Early identification and targeted interventions could improve outcomes and reduce morbidity and mortality associated with stroke in HIV-infected individuals.

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INTRODUCTION

Stroke is a neurological disorder characterized by the blockage of blood vessels (Caplan et al., 2023; Chung, 2017; Hu et al., 2017; Murphy & Werring, 2020, 2023; Olmosovna et al., 2024; Parmar, 2018). The highest reported incidence of stroke occurs in China, with an estimated 331–378 cases per 100,000 life years. The second highest rate occurs in Eastern Europe (181–218 per 100,000 life years), and the lowest in Latin America (85–100 per 100,000 life years) (Johnson et al., 2019). HIV infection can cause stroke through several mechanisms, including opportunistic infections, vasculopathy, cardioembolism, and coagulopathy (Bozzette, 2011; Musuka et al., 2015; Roger et al., 2011; Wang et al., 2010).

However, stroke and HIV infection often coexist. Between 1% and 5% of patients with HIV experience stroke in clinical settings, although a higher proportion (4–34%) have cerebral ischemic lesions at autopsy (Azizah & Wahyuningsih, 2020; Murphy & Werring, 2020). There is little correlation between pathological evidence of cerebral ischemic lesions and clinical manifestations before death in series assessing this relationship. In the United States,

hospitalizations for patients with stroke and concomitant HIV infection increased by 43% over nine years (Wang et al., 2010).

Despite this apparent association, surprisingly few studies have assessed the impact of HIV infection on the burden and nature of stroke—such as the extent to which HIV increases stroke risk and the underlying pathogenesis of stroke in individuals with HIV. Management and prevention of stroke should include identifying and treating specific causes of stroke and associated risk factors, as well as judicious adjustment of cART regimens.

Previous studies have highlighted the intersection between HIV infection and stroke, but important gaps remain in understanding their clinical interplay. For example, Benjamin et al. (2012) examined the epidemiology of stroke in HIV-positive populations in the United States, finding that HIV infection increases the risk of ischemic stroke through mechanisms such as vasculopathy and coagulopathy. However, their study primarily focused on large-scale epidemiological data and did not assess individual clinical manifestations, risk factors, or stroke subtypes in depth. Similarly, Shah et al. (2018) analyzed cerebrovascular events in HIV-infected patients, emphasizing that opportunistic infections and antiretroviral therapy can contribute to stroke occurrence, yet their study lacked detailed case-level analysis linking HIV-related factors to acute stroke presentations and clinical outcomes.

These limitations highlight a gap in the literature regarding comprehensive, clinically detailed evaluations of stroke in HIV-positive patients, particularly case-based insights integrating pathophysiology, risk factors, and clinical presentation. The purpose of this research is to provide a nuanced understanding of how HIV infection may exacerbate cerebrovascular risk and influence stroke presentation. The benefits include informing clinicians about potential cerebrovascular complications in HIV-positive patients, guiding risk assessment and prevention strategies, and contributing empirical evidence to a relatively underexplored area of HIV-related neurological outcomes. By integrating clinical, pathological, and therapeutic considerations, this study provides valuable insights for both individualized patient care and broader research on stroke in HIV populations.

METHOD

A 56-year-old man came with complaints of weakness in the left extremity. Weakness in the left extremity was felt since 1 day ago. The patient could not eat and drink, accompanied by nausea, vomiting 1 time containing fluid. There is a history of stroke 5 years ago and 4 years ago, a history of HIV 2 years ago. On physical examination, somnolent consciousness was found, GCS 9 (E3V1M6), blood pressure 172/111, pulse rate 81 beats / minute, respiratory rate 22 breaths / minute, body temperature 37.2 C. On neurological examination, neurological deficits were found in the facial nerve, vagus nerve, hypoglossal nerve, motor weakness of the left side of the upper and lower extremities (motor strength 1), Babinski reflex - / +. A CT scan of the head on June 29, 2025 showed ICH in the right basal ganglia with a volume of 32.5 cc accompanied by perifocal edema that pressed and narrowed the right lateral ventricle, IVH in bilateral lateral ventricles, III and IV (Figure 1). Management was given 200 cc of mannitol loading, followed by 6x100 taper-off therapy, administration of 2x500 mg citicoline injections, 500 mcg mecobalamin, 2x1 ranitidine, 3x1 antrain, 3x250 mg glauseta.

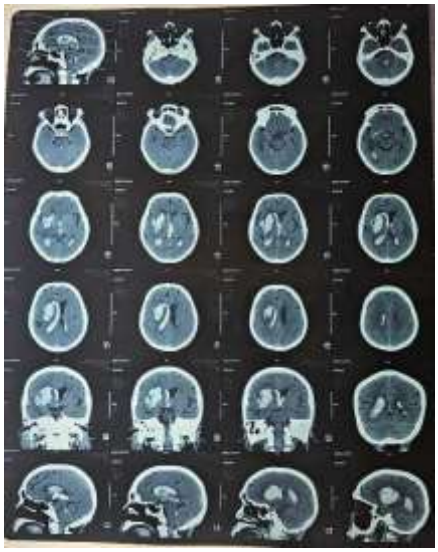

Figure 1. ICH in the right basal ganglia with a volume of approximately 32.5 cc accompanied by perifocal edema compressing and narrowing the right lateral ventricle, IVH in bilateral lateral ventricles, III and IV

RESULT AND DISCUSSION

Stroke is a neurological disorder characterized by the blockage of a blood vessel. Blood clots form in the brain and disrupt blood flow, block arteries, and cause blood vessels to rupture, resulting in bleeding. The rupture of arteries leading to the brain during a stroke results in the sudden death of brain cells due to oxygen deprivation. Stroke can also cause depression and dementia. In some cases, neurological deficits and weakness on one side of the body suggest a diagnosis of stroke.

Stroke is the second leading cause of death worldwide. Stroke affects approximately 13.7 million people and kills approximately 5.5 million each year. Approximately 87% of strokes are ischemic infarctions, a prevalence that increased substantially between 1990 and 2016, driven by decreased mortality and improved clinical interventions. Primary (first-time) hemorrhage accounts for the majority of strokes, with secondary (second-time) hemorrhages estimated to account for 10–25% (Johnson et al., 2019; Roger et al., 2011). A head CT scan revealed intracranial hemorrhage in the right basal ganglia, causing brain edema and decreased consciousness.

Stroke is defined as a sudden neurological explosion caused by impaired perfusion through blood vessels to the brain. Blood flow to the brain is regulated by the two internal carotid arteries anteriorly and the two vertebral arteries posteriorly (circle of Willis). Ischemic strokes are caused by insufficient blood and oxygen supply to the brain; hemorrhagic strokes are caused by bleeding or leaking blood vessels. Ischemic occlusions contribute to approximately 85% of stroke deaths, with the remainder caused by intracerebral hemorrhages. Ischemic occlusions result in thrombotic and embolic events in the brain (Musuka et al., 2015).

Hemorrhagic strokes account for approximately 10–15% of all strokes and have a high mortality rate. In this condition, stress on brain tissue and internal injury cause blood vessels

to rupture. This has a toxic effect on the vascular system, resulting in an infarction. Hemorrhagic strokes are classified into intracerebral hemorrhage and subarachnoid hemorrhage. In ICH, a blood vessel ruptures, causing an abnormal accumulation of blood within the brain. The main causes of ICH are hypertension, vascular disorders, and excessive use of anticoagulants and thrombolytic agents. In subarachnoid hemorrhage, blood accumulates in the subarachnoid space of the brain due to head injury or cerebral aneurysm (Aronowski & Zhao, 2011). The risk of stroke increases with age and doubles in both men and women over the age of 55. In one case, the risk factor was 56 years old.

Stroke prevention involves modifying risk factors in a population or individual, while stroke management depends on addressing the pathophysiology. Treatment includes administering mannitol to reduce brain edema and administering nootropics and neuroprotectants.

HIV Infection and Stroke

HIV infection can cause stroke through several mechanisms, including opportunistic infections, vasculopathy, cardioembolism, and coagulopathy. However, stroke and HIV infection often coexist. HIV-associated vasculopathy describes a variety of cerebrovascular changes, including stenosis and aneurysm formation, vasculitis, and accelerated atherosclerosis, and may be caused directly or indirectly by HIV infection, although the mechanisms remain controversial. The mechanisms are specific to atheroma and potentially apply to other forms of HIV-associated vasculopathy. (A) Direct damage may occur through persistent exposure of the endothelium to HIV virions or viral particles (e.g., GP120 or TAT), leading to endothelial dysfunction. (B) Indirect damage may arise from circulating infected monocytes freely transmigrating to the endothelium as part of normal surveillance, with impaired reverse transmigration, thus increasing the subendothelial population of HIV-infected monocytes. Release of chemokines such as CCL2 from infected leukocytes attracts more leukocytes. (C)

Several events contribute to the development of damage and the spread of atherogenesis: upregulation of cell adhesion molecules (e.g., selectins), leading to increased adhesion of infected and uninfected leukocytes; release of HIV virions into arterial smooth muscle and continued active viral replication within smooth muscle cells; release of inflammatory cytokines from HIV-infected cells, leading to further leukocyte recruitment and adhesion, increased production of reactive oxygen species, and disruption of the vascular wall.

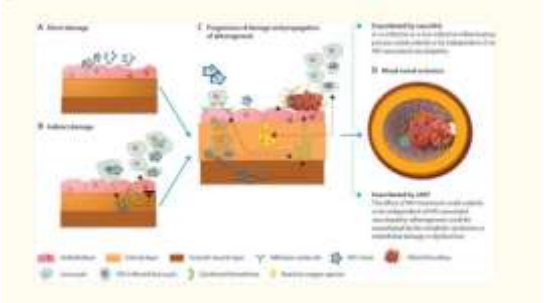

Figure 2. Pathogenesis of vasculopathy and atherogenesis in HIV

In resource-limited settings, investigation and treatment should be directed toward identifying treatable or stroke-like causes of stroke, such as tuberculosis, cryptococcus, toxoplasmosis, varicella zoster, or herpesvirus infection, possibly by combining CT brain scanning, chest radiography, lumbar puncture (if not contraindicated and in the absence of an alternative cause—for example, a cardioembolic source), and specific blood tests (Figure 3). Additional tests may be necessary to determine the cause of the stroke (for example, sputum and CSF samples for tuberculosis). Intrathecal IgG measurement to varicella zoster virus along with CSF DNA PCR increases the likelihood of identifying these potential causes. The diagnosis of neurosyphilis in patients with HIV can be complicated (Hasan & Adisasmito, 2017). A positive CSF venereal disease laboratory test can be helpful—when this test is negative in patients with HIV, CSF treponemal antibody testing appears to be a reasonable approach. Antineutrophil cytoplasmic antibodies (ANCA) as assessed by immunofluorescence and enzyme-linked immunosorbent assay have been identified in patients with HIV, but not necessarily in patients with vasculitis, autoimmune disease, or specific opportunistic infections (Costa et al., 2017). The diagnosis of cerebral vasculitis in patients with HIV should be based on appropriate radiological findings and, if possible, histologic findings, in the absence of other potential causes of vasculitis (e.g., opportunistic infections). In these circumstances, detection of ANCA can confirm the diagnosis.

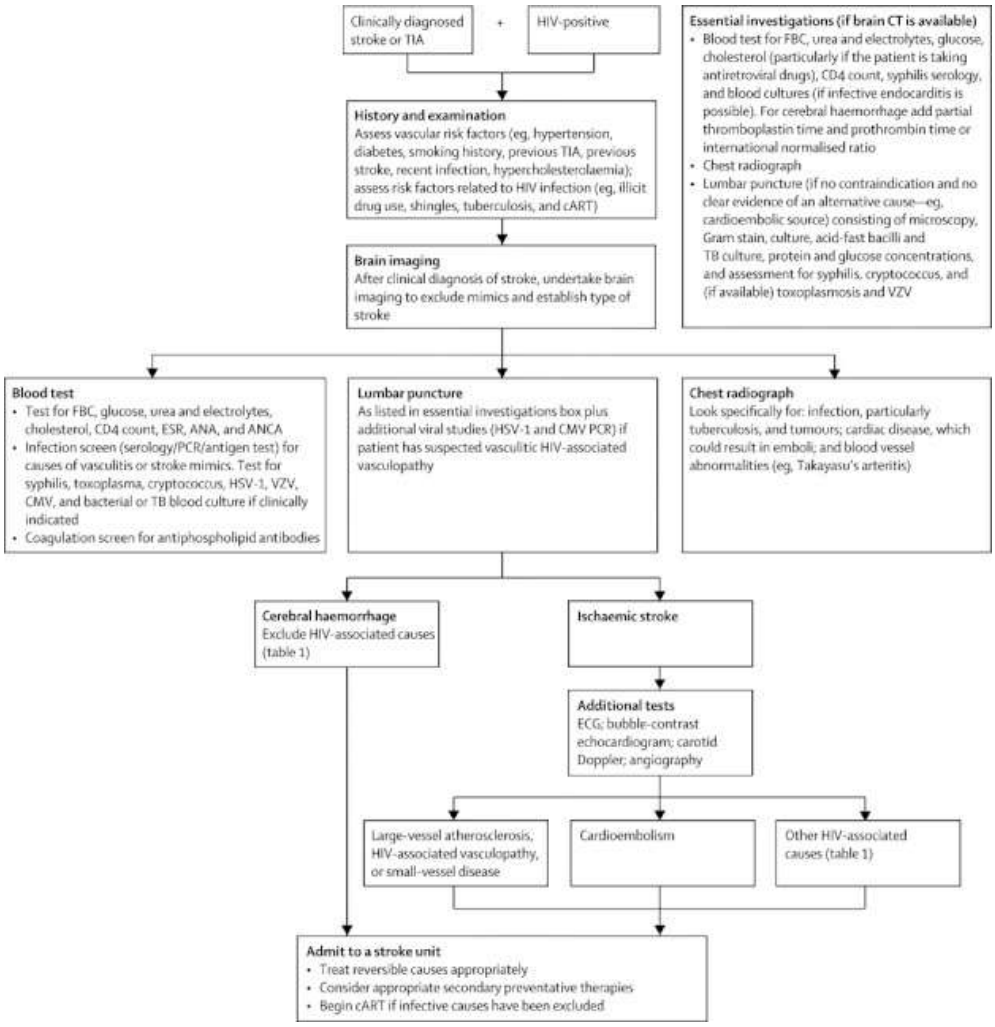

Figure 3. Management approach for HIV-infected patients with stroke. cART: combined antiretroviral therapy

The role of immunosuppression with corticosteroids remains unclear. In the absence of evidence to guide management, it seems reasonable to initiate cART (with corticosteroids if the patient has a poor response) if HIV-related vasculopathy is suspected and other autoimmune or infectious causes have been ruled out. Evidence has convincingly demonstrated that cART results in reduced all-cause mortality in patients with HIV. Much less certain is whether cART treatment, particularly exposure to protease inhibitors, increases the long-term risk of stroke and myocardial infarction due to metabolic effects (e.g., hypercholesterolemia, as already described) and prolonged survival (aging is a risk factor for stroke, and some HIV-infected populations have a high prevalence of smoking) (Bozzette et al., 2011). The risk-benefit ratio of cART, based on current knowledge, appears favorable. However, given concerns about long-term stroke and cardiovascular disease risk, a pragmatic approach seems reasonable—that is, clinicians should identify and manage risk factors, perhaps changing the class of cART regimen, or considering cholesterol-lowering medications if needed. No studies have guided the use of secondary prevention for stroke, including the use of antiplatelet, statin, and blood pressure-lowering therapy, which are directly applicable to patients with HIV who have had a

stroke. (Aronowski & Zhao, 2011; Jones et al., 2020; Shurtleff & Lawrence, 2012; Society, 2018) However, general lifestyle factors and reduction of vascular risk factors seem reasonable. Finally, the patient's relevant mode of HIV infection should be considered, as this may influence underlying stroke risk factors, etiology, and management. In Sub-Saharan Africa, the primary mode of HIV transmission is heterosexual sexual intercourse, while transmission through IDUs is rare (Wamai et al., 2011). However, a history of IDU use is relevant, especially in areas where it is common, as it may be associated with several potential causes of stroke, including the use of certain drugs (cocaine, amphetamines, sympathomimetic drugs), infective endocarditis, and particulate matter embolization (Warlow C, van Gijn J, Dennis M. Stroke: practical management. Blackwell Publishing; Malden, MA: 2008 et al., 2011). In many regions, smoking is more common in people with HIV than in the general population (Shurtleff et al., 2012).

CONCLUSION

This case report demonstrates that patients with HIV infection are at risk for stroke due to various mechanisms, including the direct effect of HIV on the vascular endothelium, immunodeficiency, opportunistic infections, the use of certain antiretroviral therapies, and the presence of traditional cardiovascular risk factors. Early diagnosis, evaluation of comorbid risk factors, and comprehensive management are crucial to reducing morbidity and mortality in HIV-infected patients with stroke. In HIV-infected patients, routine screening for cardiovascular risk factors and monitoring for the side effects of antiretroviral therapy are essential to prevent cerebrovascular events. Further research is needed on the relationship between HIV, ART regimens, and stroke to strengthen the scientific evidence and formulate more effective prevention protocols.

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