

Cerebral Aneurysmal Vasculopathy Presenting as Central Facial Palsy in a 25-Year-Old Male with Acquired Immunodeficiency Syndrome

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Abstract.

Introduction. Intracranial aneurysms have a global prevalence of ~2.3%, but the Human Immunodeficiency Virus (HIV)-related proportion is unclear. Acquired Immunodeficiency Syndrome (AIDS) can cause cerebral vasculopathy via direct viral endothelial injury or indirect mechanisms such as cardioembolism, coagulopathy, or non-HIV-related vasculitis. HIV-associated vasculopathy involves intracranial or extracranial vessel abnormalities unrelated to opportunistic infections or neoplasms.

Case Presentation. A 25-year-old male with AIDS, poorly compliant with efavirenz/lamivudine/tenofovir, presented with a 2-week history of facial palsy, initially treated as Bell's palsy with prednisone. After seven days, he developed left hemiparesis, expressive aphasia, and abulia. MRI/MRA revealed fusiform and saccular aneurysms in the middle, anterior, and posterior cerebral arteries, with acute, subacute, and chronic infarcts. Cardioembolic and carotid causes were excluded. Due to high rupture risk, no surgical intervention was performed. Highly active antiretroviral therapy (HAART) was switched to dolutegravir/lamivudine/tenofovir, alongside antifungal therapy, PCP prophylaxis, and occupational therapy. At six-month follow-up, the patient remained HAART-compliant, ambulated independently, and continued rehabilitation; expressive aphasia persisted.

Conclusion. HIV-associated cerebral vasculopathy should be considered in young AIDS patients with new focal deficits, including facial palsy. Early HAART may improve short-term outcomes.

Keywords. HIV cerebral vasculopathy, AIDS, HAART, case report

Introduction

Intracranial aneurysms are uncommon, with an estimated global prevalence of 2.3%, though the proportion attributable to HIV remains unclear¹. HIV-associated cerebral aneurysmal vasculopathy is rare, first described in pediatric patients in 1989 and increasingly recognized in adults since 2006². The local incidence is unknown, and to our knowledge, this is the first documented case in our setting.

The pathogenesis is multifactorial, involving direct viral-mediated endothelial injury, opportunistic infections, chronic systemic inflammation, and immune reconstitution inflammatory syndrome (IRIS)³⁻⁶. HIV can damage vessel walls through viral proteins, circulating infected monocytes, and inflammatory cytokines, while co-infections may further weaken vascular integrity by

releasing elastase and degrading the internal elastic lamina⁷⁻¹⁰. These mechanisms lead to fusiform or saccular aneurysm formation, with the middle cerebral artery most commonly affected, followed by the anterior cerebral artery³.

Clinically, patients often present with focal neurologic deficits, ischemic infarction, or subarachnoid hemorrhage^{3, 4}. Most have advanced HIV with low CD4 counts, though aneurysms have also been reported after HAART initiation, suggesting a possible role for IRIS^{2,3}. Neurologic presentations may mimic more common disorders, delaying recognition.

Given the rarity and potential severity of HIV-associated cerebral vasculopathy, early neuroimaging and prompt HAART initiation may influence prognosis^{2, 3, 7}. This report describes a unique case initially manifesting as central facial palsy in a young adult with AIDS, underscoring the need for heightened clinical suspicion in atypical neurologic presentations.

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Case Presentation

A 25-year-old male with AIDS presented with a two-week history of left facial palsy. Initially diagnosed as Bell's palsy by a local physician, he was given prednisone 60 mg daily for seven days and vitamin B complex. One week later, he developed left-sided hemiparesis, abulia, and dysphagia. There was no fever, headache, loss of consciousness, or seizure.

Past medical history included AIDS diagnosed six months earlier (latest CD4 count 53 cells/mm³, one month before admission), with poor compliance to HAART - efavirenz/lamivudine/tenofovir. He had tuberculous lymphadenitis treated with isoniazid/rifampicin/pyrazinamide/ethambutol (HRZE) for two months, followed by isoniazid/rifampicin (HR) for four months, and one month of trimethoprim-sulfamethoxazole prophylaxis. Hepatitis B surface antigen and rapid plasma reagin were negative. There was no family history of aneurysmal disease or similar neurologic conditions. He denied hypertension, diabetes, smoking, alcohol, or illicit drug use.

On examination, he was cachectic with bitemporal wasting. Vitals: BP 100/80 mmHg, HR 110 bpm, RR 21/min, Temp 36.8 °C, O₂ saturation 97%. There were generalized hyperpigmented macular rashes and oral plaques. He was alert, oriented, communicated in writing, and had left facial asymmetry with impaired tongue protrusion. Remaining cranial nerves were intact. Motor strength was 5/5 on the right and 4/5 on the left; sensation was intact. Babinski sign was negative; cerebellar signs were absent.

An NGT was inserted for feeding. Due to oral thrush, he was started on fluconazole 200 mg daily for 14 days, trimethoprim-sulfamethoxazole 960 mg daily for PCP prophylaxis, and citicoline 1 g daily.

Given his acute focal neurologic deficits, differential diagnoses included Bell's palsy, cardioembolic stroke, infectious vasculitis, and hypercoagulable state. Laboratory work-up revealed leukocytosis (WBC 18.4 × 10³/μL) with lymphocytic predominance. Chest X-ray was unremarkable. A 12-lead ECG showed sinus tachycardia without arrhythmia. Transthoracic echocardiography demonstrated normal left ventricular geometry and contractility with no valvular vegetations, making

cardioembolic stroke unlikely. Carotid duplex revealed normal antegrade flow in both vertebral arteries, excluding significant extracranial stenosis. Stroke-in-the-young work-up, including Protein C and Protein S levels, antiphospholipid antibodies, and Factor V Leiden mutation testing, was not performed due to financial constraints.

MRI with MRA of the brain (Figures 2-5) demonstrated acute infarcts in the left semi-oval center, left corona radiata (Fig. 2), and right cerebral peduncle; a subacute infarct in the right basal ganglia (Fig. 2); and a chronic infarct in the left basal ganglia. Hemosiderin deposits were noted along the left sylvian fissure and left frontal sulci, with a few non-specific hyperintensities in the right thalamus and the genu of the left internal capsule. There was fusiform aneurysmal dilatation of the M1 segment of the left MCA and multiple saccular aneurysms involving both MCAs and the left ACA. Fusiform aneurysmal dilatation was also seen in the A1 segment of the left ACA and the anterior communicating artery, with the latter measuring approximately 1.8 × 1.3 cm in longest axial by perpendicular diameter (Fig. 3 and Fig. 5). Additional fusiform dilatation was present in the left posterior cerebral artery. No intraluminal filling defect or signs of venous thrombosis were observed.

The pattern of vascular changes, combined with advanced immunosuppression, supported a diagnosis of HIV-associated cerebral aneurysmal vasculopathy. Neurosurgery advised against aneurysm repair due to the high rupture risk, and lumbar puncture was deferred for the same reason.

On hospital day two, HAART was changed to dolutegravir/lamivudine/tenofovir once daily. PCP prophylaxis, antifungal therapy, and occupational therapy for orofacial and swallowing rehabilitation were continued.

After four days, the patient showed improvement in facial symmetry, motor strength, and speech initiation. At six-month follow-up, he remained clinically stable and reported adherence to HAART, ambulated independently, and continued occupational therapy, although expressive aphasia persisted.

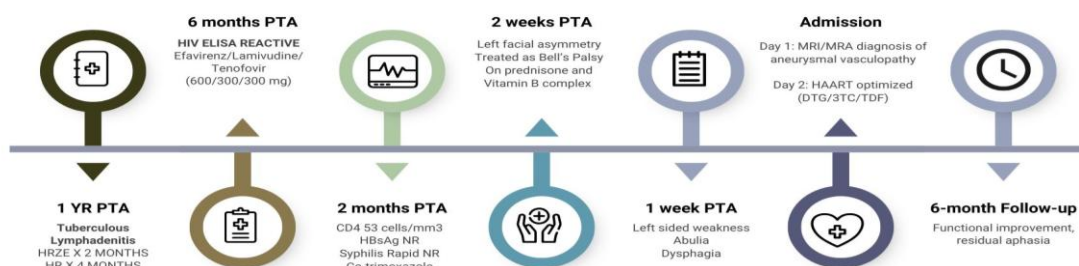


Figure 1. Timeline of symptoms and clinical course

The timeline summarizes the patient's clinical history from one year prior to admission, including treatment for tuberculous lymphadenitis, diagnosis of HIV infection and initiation of antiretroviral therapy, progression to severe immunosuppression, and the onset of focal neurologic symptoms leading to hospital admission.

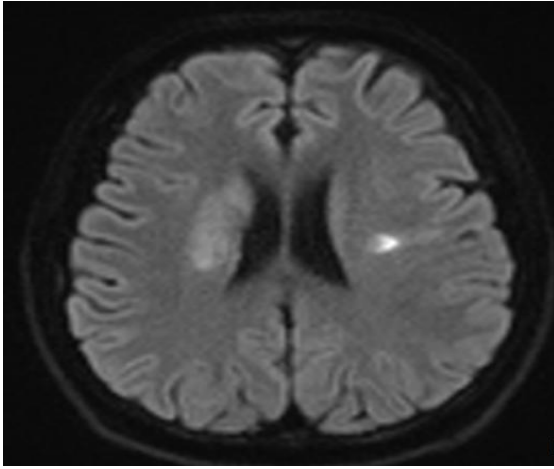


Figure 2. MRI FLAIR image of acute infarct in the left corona radiata and subacute infarct in the right basal ganglia

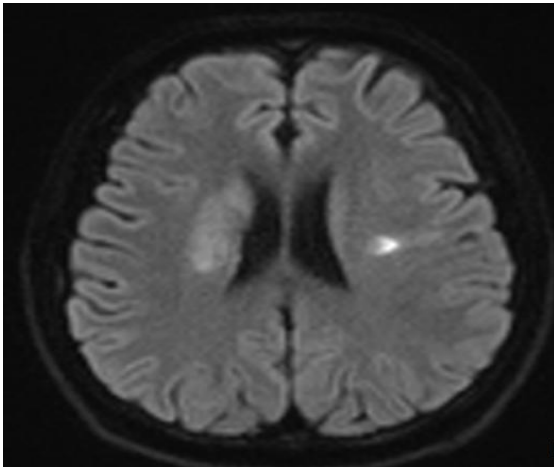


Figure 3. MRI FLAIR image of fusiform aneurysm of Anterior Communicating Artery

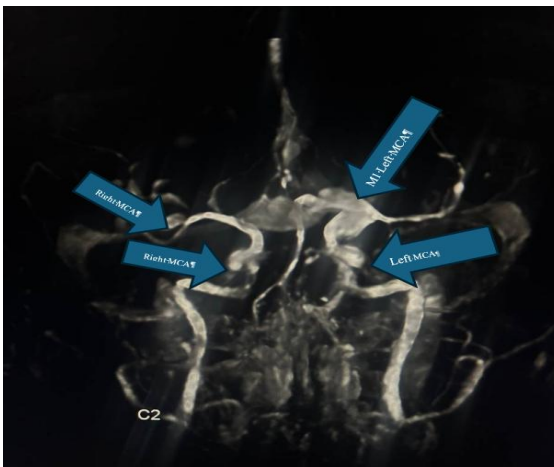


Figure 4. MRA image of saccular aneurysms in middle cerebral arteries (MCA) and fusiform aneurysms in M1 of MCA

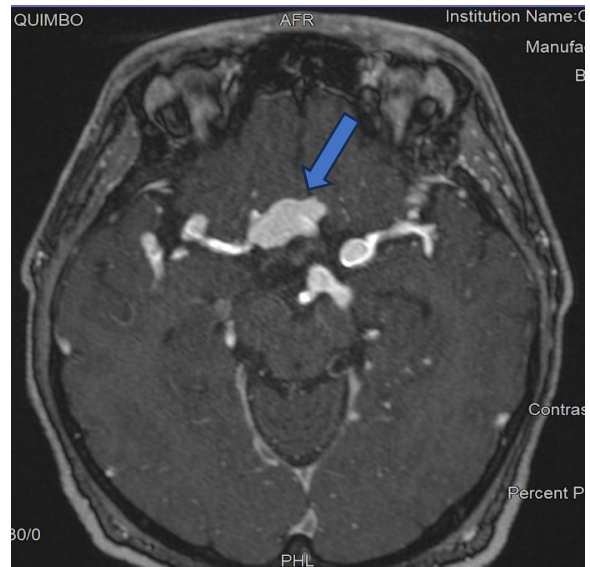


Figure 5. MRI FLAIR image of acute infarct in the left corona radiata and subacute infarct in the right basal ganglia

Case Discussion

HIV-associated vasculopathy can predispose to cerebral aneurysm development through an inflammatory process wherein HIV viral proteins lead to increased chemoattractant and adhesion molecules in the vessel wall¹⁰. Diffuse intracranial aneurysmal vasculopathy affects young adults with significant immunocompromise from HIV disease^{2,3}. These patients tend to have CD4 counts <200 and high HIV viral loads^{3,4}. Radiographic findings are characteristic diffuse fusiform aneurysms, often with hemorrhage or infarct^{3,7}. In this case, the patient had a CD4 count of 53 cells/mm³ and had saccular and fusiform aneurysms involving the anterior and posterior circulation with areas of infarcts. Eleven published and one unpublished adult case of HIV-related intracranial fusiform aneurysmal vasculopathy showed that one of the twelve reported adult cases underwent autopsy and demonstrated fusiform dilatation of the left internal carotid artery and middle cerebral artery with luminal thrombosis, concentric intimal fibrosis, atrophic media, and elastic lamina thinning and fragmentation⁷⁻¹⁰.

A systematic review on HIV-associated cerebral aneurysmal vasculopathy by Baeesa et al.³ showed that the age of the adult patients with this condition ranged from 20 to 54 years with a median of 36.5 years. Male patients accounted for 56.3% of the reported cases. As with this case report, the patient is a 25-year-old male. The mean duration of infection before the diagnosis of the aneurysm was 4.6 years (ranging from 0-19 years). For this patient, he was diagnosed six months prior to admission³.

Weakness was the most common presenting symptom among adult patients (50%), followed by headache (31.3%), confusion (25%), and speech disorders (25%)³. As for this patient, he initially had facial palsy which

progressed to hemiparesis, expressive aphasia, and abulia. Thirty-seven point five percent of adult patients had ischemic cerebral infarction at the time of aneurysm diagnosis³. HIV infection appears to increase the risk of both cerebral ischemia and hemorrhage^{5,6}. Moreover, hemorrhagic ischemia was reported in 43.8% of adult patients³. This may explain the weakness that most reported patients presented with. As in our case, areas of acute infarcts were noted in the left semioval center, left corona radiata, and right cerebral peduncle; subacute infarct in the right basal ganglia and chronic infarct in the left basal ganglia. The bilaterality of infarcts explains the patient's symptoms, including difficulty opening the mouth and limitations in protruding the tongue.

In the same study by Baeesa et al.³, most of the aneurysms were multiple (93.8% of adult cases). Fusiform aneurysms were reported in 75% of adult cases, while saccular aneurysms were reported in 12.5% of cases. One adult case had both saccular and fusiform aneurysms. As for our patient, he had both saccular and fusiform aneurysms, making this case particularly rare.

Because of the rarity of reports of this condition in the literature, optimal management is not yet clear^{3,7}. Response to highly active antiretroviral therapy (HAART) varied in literature, with some reports demonstrating survival benefits when HAART is initiated early^{3,7}. Surgical or endovascular management of ruptured or progressing HIV-associated cerebral aneurysms is recommended regardless of HAART^{3,7,8}. In our case, from a neurosurgical standpoint, the risk of rupture was significant, making surgical intervention for aneurysm reconstruction too risky. As a result, no surgical management options were pursued for the aneurysms.

In a study by Kyle et al.², two cases of HIV-associated vasculopathy with features of fusiform aneurysm in uncontrolled HIV were presented. One case described a 52-year-old woman with a 25-year history of HIV, diagnosed with anterior fusiform aneurysms after 18 years. She continued HAART and remained neurologically stable for over 15 years, without further infarcts or cognitive decline; her most recent HIV viral load was undetectable and her CD4 count was 516 cells/mm³. The second case described a 48-year-old man with a CD4 count of 8 cells/mm³ who presented with cerebral toxoplasmosis after four years of HAART non-compliance. Upon reinitiation of HAART and trimethoprim-sulfamethoxazole, repeat CT angiography nine months later revealed decreased size of the fusiform dilatations. Improvement in HIV control was linked to long-term stability in Case 1 and an actual reduction in aneurysm size in Case 2². As in our patient, he was discharged on HAART comprising dolutegravir/lamivudine/tenofovir. After a six-month follow-up, the patient has remained compliant with his HAART regimen and has continued occupational therapy. He is now able to ambulate unassisted; however, expressive aphasia persists. A repeat MRI was requested to assess aneurysm status, but this was not performed due to financial constraints.

The strengths of this case include the rare presentation of HIV-associated cerebral aneurysmal vasculopathy manifesting initially as central facial palsy, detailed neuroimaging findings involving both anterior and posterior circulation, and a multidisciplinary management approach. Limitations include the inherent constraints of a single-case design, limited duration of follow-up, lack of serial neuroimaging, and reliance on patient-reported adherence to antiretroviral therapy due to resource limitations.

Patient's Perspective

When I first became ill, I constantly felt tired and experienced persistent fever, headaches, and noticeable weight loss. I also had swelling of the lymph nodes in my neck. The most frightening part of my illness occurred when I became paralyzed for two days and later suffered a stroke, after which I was diagnosed with aneurysm. During treatment, I noticed significant weight gain due to increased appetite and experienced changes in my mood, including a shorter temper. Despite these challenges, I am now doing better and continuing my medications as prescribed. Through this experience, I realized how difficult illness can be, and I believe prevention is very important. I strongly encourage others to practice safe sex, as prevention is always better than cure.

Conclusion

HIV-associated cerebral vasculopathy is a rare but serious cerebrovascular complication of HIV infection. It most often presents with weakness and other focal neurological deficits, including speech disorders. Severe immunosuppression, as well as the potential presence of secondary opportunistic infections that can also compromise cerebral vasculature, may obscure or delay diagnosis. Clinicians should maintain a high index of suspicion and promptly consider neuroimaging when evaluating HIV patients with new neurological deficits. Although optimal management remains undefined due to the rarity of this condition, several reports suggest that early initiation or optimization of may improve outcomes and, in some cases, stabilize or reduce aneurysmal changes. Early recognition and timely intervention therefore remain essential to improving prognosis in affected patients.

Recommendation

1. Conduct systematic data collection to establish the local incidence and prevalence of cerebral vasculopathy among patients with AIDS, which will aid in understanding its burden and guiding resource allocation.
2. Implement prospective, longitudinal studies to evaluate clinical, functional, and radiologic outcomes in AIDS patients with cerebral vasculopathy managed with

dolutegravir/lamivudine/tenofovir, assessing both neurological recovery and vascular stability.

3. Monitor and integrate findings from large-scale randomized controlled trials, such as the REPRIEVE trial, to inform strategies for preventing vascular events in HIV-infected individuals and to adapt these insights to the local clinical context

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