Journal of AIDS and HIV Infections
Volume 11 | Issue 1
2454-499X

Case Report Open Access

HIV-Associated Intracranial Aneurysmal Vasculopathy in Adults: A Rare but Devastating Neurological Complication

Kamalesh Tayade*, Anarya Karle, Haresh Bharote, Dnyaneshwar Asole, Anil Venkitachalam, Amarnath Chavan and Jivan Morey

Department of Neurology, LTMMC and GH, Mumbai, India

Corresponding Author: Kamalesh Tayade, Department of Neurology LTMMC and GH, Mumbai, India, Email: drkamalesh21@gmail.com

Citation: Kamalesh Tayade, Anarya Karle, Haresh Bharote, Dnyaneshwar Asole, Anil Venkitachalam, et al. (2026) HIV-Associated Intracranial Aneurysmal Vasculopathy in Adults: A Rare but Devastating Neurological Complication. J Aids Hiv Inf 11(1): 102.

Received Date: May 05, 2026 **Accepted Date:** May 15, 2026 **Published Date:** May 22, 2026

Abstract

Introduction: HIV-associated intracranial aneurysmal vasculopathy is a rare, poorly understood, and almost invariably catastrophic complication of advanced immunosuppression. It's defining feature — multiple fusiform aneurysms erupting along the walls of cerebral arteries — reflects a convergence of viral endothelial injury, chronic inflammation, and opportunistic pathogen invasion that together dismantle the structural integrity of the cerebrovascular tree.

Case report: We present a 40-year-old woman with newly diagnosed HIV (CD4 count 114 cells/mm^3) who deteriorated sharply two weeks after antiretroviral therapy initiation, presenting with seizures, altered consciousness, and right hemiparesis. Digital subtraction angiography revealed bilateral fusiform middle cerebral artery aneurysms with the characteristic beading pattern of HIV vasculopathy. Cerebrospinal fluid confirmed concurrent fulminant cryptococcal meningitis. Despite maximal therapy, she died on day four from refractory intracranial hypertension and brainstem herniation.

Conclusion: Clinicians practising in HIV-endemic regions must keep this diagnosis in sharp focus when a young patient presents with cerebrovascular disease and no conventional risk factors. Early vascular imaging is essential; prognosis below a CD4 count of 200 cells/mm³ remains dismal.

Keywords: HIV-Associated Vasculopathy; Intracranial Aneurysms; Cerebrovascular Disease; Digital Subtraction Angiography; Subarachnoid Haemorrhage; Opportunistic Infections

Introduction

HIV is no longer understood purely as a disease of immune failure. Decades of clinical observation have established that uncontrolled viral replication causes damage well beyond the CD4 compartment, affecting the cardiovascular system, kidneys, liver, and central nervous system in ways that are sometimes more immediately life-threatening than immunosuppression itself. Among the less commonly recognised manifestations is its capacity to damage the walls of cerebral arteries to a degree that results in aneurysm formation — a complication that is difficult to diagnose, rarely reversible, and associated with high mortality [1].

HIV-infected individuals have approximately twice the stroke risk of age-matched HIV-negative controls [2]. The mechanisms are multiple: accelerated atherosclerosis driven by chronic systemic inflammation, cardioembolism from HIV-associated cardiomyopathy, antiphospholipid antibody-related coagulopathy, and direct arterial wall injury [1,3].What separates HIV-associated intracranial aneurysmal vasculopathy from other HIV-related cerebrovascular disease is its distinctive morphology. These are not the saccular aneurysms that form at bifurcation points and are familiar to most neurologists and neurosurgeons [1,4,5]. They are fusiform — involving the full circumference of the vessel over a longitudinal segment — and frequently alternate with zones of focal narrowing along the same artery, producing the beading appearance that is characteristic on angiography neurosurgeons [1,4,5].

The pathogenesis involves several concurrent mechanisms. HIV-1 gains access to cerebrovascular endothelial cells through CCR5 and CXCR4 co-receptors, independent of the classical CD4-mediated entry route. Once inside, viral proteins — principally gp120, Tat, and Nef — disrupt endothelial barrier function, upregulate inflammatory adhesion molecules, stimulate cytokine release, and activate matrix metalloproteinases that break down the collagen and elastin fibres responsible for arterial wall tensile strength [3]. The result is a vessel wall that is chronically inflamed and structurally compromised. Opportunistic pathogens add further injury on top of this baseline. Varicella-zoster virus can establish granulomatous arteritis within the vessel wall itself, while Cryptococcus neoformans extends from infected subarachnoid tissue directly into the arterial adventitia, causing inflammatory destruction of the tunica media that significantly worsens aneurysm formation when superimposed on pre-existing HIV-mediated endothelial damage [6,7].

A clinically important and somewhat counterintuitive contributor is immune reconstitution inflammatory syndrome. When antiretroviral therapy is initiated in a severely immunosuppressed patient, the recovering immune system can mount a dysregulated inflammatory response against residual pathogen antigens within vessel walls. Cases have been documented in which new or enlarging intracranial aneurysms appeared within weeks of ART initiation, suggesting that a subset of patients may experience accelerated vascular injury at precisely the point when treatment begins [8].

Case Presentation

A 40-year-old woman arrived at our neurological intensive care unit acutely unwell. She had experienced two generalized tonic-clonic seizures the preceding day, was progressively difficult to arouse, and her right arm and leg had been weakening for a week. Her history, reconstructed from family and referral records, was a sequence of compounding misfortune: pulmonary tuberculosis diagnosed one year prior, abandoned after only 40 days of therapy, followed by a new admission with tuberculous meningitis at a district hospital where HIV was discovered for the first time. Antiretroviral therapy had been commenced just two weeks before this deterioration.

On examination, she was drowsy with a Glasgow Coma Scale score of 11 out of 15. Blood pressure was 130/80 mmHg, temperature 38.4°C. There was a left gaze preference, sluggish bilateral pupils, and right-sided hemiplegia with power graded 2 out of 5 in the upper limb and 1 out of 5 in the lower limb. Bilateral crepitations were present on chest auscultation.

Laboratory investigations confirmed severe immunosuppression: CD4 count 114 cells/mm^3, plasma viral load 186,000 copies/mL, haemoglobin 10.7 g/dL, erythrocyte sedimentation rate 56 mm/hour, and serum albumin 2.8 g/dL. Cerebrospinal fluid examination was decisive — opening pressure was elevated at 28 cm H_2O_2 with lymphocytic pleocytosis (80 cells/mm^3, 78% lymphocytes), protein 125 mg/dL, glucose 38 mg/dL, and a CSF-to-serum glucose ratio of 0.37. Cryptococcal antigen was strongly positive at a titre of 1:640, and India ink preparation confirmed encapsulated yeast forms. GeneXpert for tuberculosis was negative.

MRI brain demonstrated bilateral watershed-zone ischaemic infarcts on diffusion-weighted imaging and T2-FLAIR sequences, cortical blooming artifacts on T2-star gradient echo confirming subarachnoid haemorrhage, and leptomeningeal enhancement in the basal cisterns. Digital subtraction angiography — the gold standard for intracranial vascular imaging — revealed multiple bilateral fusiform aneurysms involving M1 and M2 segments of both middle cerebral arteries, the largest measuring 8 mm, with alternating stenosis and dilatation producing the classic beading appearance throughout the anterior circulation (Figure 1).

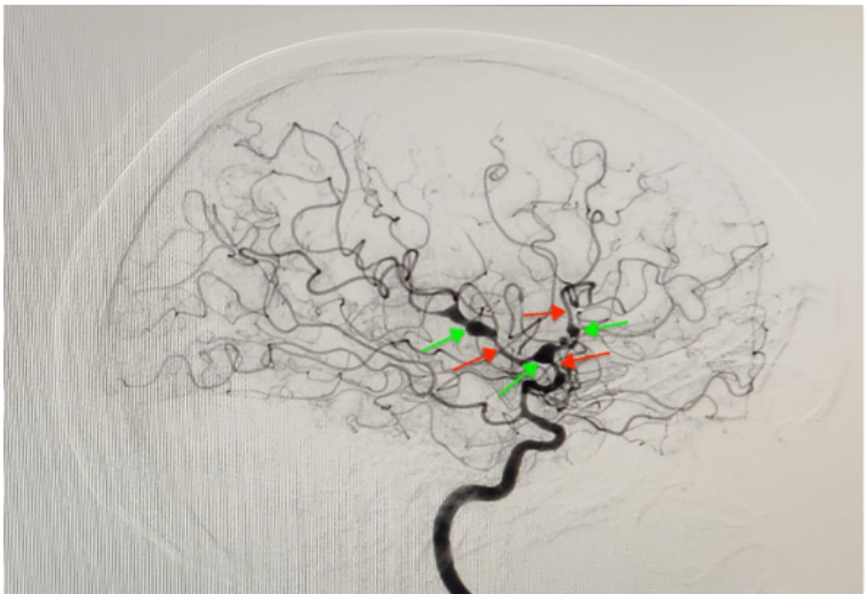

Figure 1: Cerebral DSA showing multiple fusiform aneurysm (Green arrow) and area of vessel constriction (Red arrow) in middle cerebral artery.

Management encompassed induction antifungal therapy with liposomal amphotericin B (3 mg/kg/day) plus flucytosine (100 mg/kg/day) per IDSA guidelines for HIV-associated cryptococcal meningitis, continued antiretroviral therapy, restarted anti-tubercular therapy, levetiracetam for seizure prophylaxis, and osmolar therapy for raised intracranial pressure. Despite these measures, she developed bilateral fixed dilated pupils and absent brainstem reflexes by day three. Repeat CT confirmed massive cerebral to comfort oedema with uncal herniation. Following family discussion, care was transitioned focus. She died on day four.

Discussion

HIV-associated intracranial aneurysmal vasculopathy is a rare complication predominantly affecting severely immunocompromised individuals, typically with CD4 counts below 200 cells/mm [2,3]. The outcome in this case was, in retrospect, unlikely to have been different regardless of when she arrived. By the time she reached our unit, the combination of a CD4 count of 114 cells/mm³, bilateral fusiform aneurysms, active subarachnoid haemorrhage, and fulminant cryptococcal meningitis had already placed her beyond what available therapies could realistically address. What is worth examining, however, is how she got there — and whether that trajectory could have been interrupted at an earlier point.

On comparison with previously published cases, outcome of our case is following similar trajectory [4,8,9]. Patients are typically in their third to fifth decade. Most are severely immunosuppressed at diagnosis. Outcomes are poor across the board — death or significant permanent neurological deficit is the norm, not the exception. The condition has historically been described in children, but adult cases are being reported with increasing frequency, and the clinical picture in adults appears no less severe.

The role of cryptococcal meningitis in this patient warrants specific comment. Cryptococcus neoformans is not simply an incidental finding in HIV-infected patients with neurological disease — it has a well-documented capacity to invade cerebral arterial walls by direct extension from the subarachnoid space, producing granulomatous inflammation that damages the vessel wall structurally [7]. In a patient who already has HIV-mediated endothelial dysfunction as a baseline, this additional insult to arterial wall integrity is clinically significant and likely contributed substantially to the aneurysm burden seen on her angiogram. The timing of her deterioration, occurring two weeks after ART initiation, also raises a reasonable clinical question about whether immune reconstitution inflammatory syndrome played a role. IRIS-related vascular injury has been documented in both paediatric and adult cases of HIV vasculopathy, and the temporal relationship in this patient is certainly consistent with that mechanism, even if it cannot be confirmed retrospectively [8].

From a diagnostic standpoint, the threshold for investigating HIV-associated vasculopathy needs to be low in endemic settings. A young HIV-infected patient with stroke or unexplained neurological deterioration, without hypertension, diabetes, or other conventional vascular risk factors, should prompt vascular imaging rather than a default attribution to more common diagnoses. CSF examination is essential and must include cryptococcal antigen testing, given the frequency with which cryptococcal meningitis co-exists with and contributes to the vasculopathy. MRI defines the parenchymal injury; digital subtraction angiography remains necessary for definitive characterization of aneurysm morphology and distribution [5]. Management is largely empirical — ART to suppress viral replication, targeted treatment of any identified opportunistic infection, and stepwise management of elevated intracranial pressure [7,10]. The role of corticosteroids is not established and carries real risk of further immunosuppression. Endovascular or neurosurgical intervention is generally not feasible given the fusiform, multi-segment nature of these aneurysms and the overall clinical fragility of affected patients [4].

Reported mortality across published series exceeds 50% [9], CD4 count below 200 cells/mm³, coexisting CNS infection, and haemorrhagic presentation each independently worsen outcomes; their combination, as seen here, is particularly unfavourable. The patients in the literature who have survived longer term are those who achieved durable viral suppression before cerebrovascular damage became extensive — which points, practically, to earlier HIV diagnosis and uninterrupted antiretroviral therapy as the interventions most likely to change the natural history of this condition at a population level.

Conclusion

HIV-associated intracranial aneurysmal vasculopathy is a condition where delay in diagnosis is delay in the only window available for intervention. Sustained viral suppression through early antiretroviral therapy remains the single most consequential tool clinicians possess against this uniformly devastating disease.

Declarations

Declaration of previous presentation/pre-print No pre-print available. Declaration of generative AI and AI-assisted technologies in the writing process No AI tool was used.

Acknowledgement

None

Conflict of Interest

The Author(s) declare(s) that there is no conflict of interest.

Ethical Consideration

Ethical Compliance Statement: Informed patient consent was obtained.Institutional review board or ethics committee approval not required.

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

Data Availability statement

Data will be shared on request to corresponding author.

References

1. Pillay B, Ramdial PK, Naidoo DP (2015) HIV-associated large-vessel vasculopathy: A review of the current and emerging clinicopathological spectrum in vascular surgical practice. Cardiovasc J Afr 26: 70.

2. Benjamin LA, Bryer A, Emsley HCA, Khoo S, Solomon T, et al. (2012) HIV infection and stroke: Current perspectives and future directions. Lancet Neurol 11: 878-90.

3. Chi D, Henry J, Kelley J, Thorpe R, Smith JK, et al. (2000) The effects of HIV infection on endothelial function. Endothelium 7: 223-42.

4. Modi G, Ranchod K, Modi M, Mochan A (2008) Human immunodeficiency virus associated intracranial aneurysms: Report of three adult patients with an overview of the literature. J Neurol Neurosurg Psychiatry 79: 44-6.

5. Law-ye B, Carlier RY, Richard R (2018) Considerations on the relevance of cerebral fusiform aneurysms observed during HIV infection. Clin Neuroradiol 28: 357-65

6. Nagel MA, Cohrs RJ, Mahalingam R (2008) The varicella zoster virus vasculopathies: Clinical, CSF, imaging, and virologic features. Neurology 70: 853-60.

7. Mishra AK, Arvind VH, Muliyil D (2018) Cerebrovascular injury in cryptococcal meningitis. Intl J Stroke 13: 57-5.

8. Bonkowsky JL, Christenson JC, Nixon GW, Pavia AT (2002) Cerebral aneurysms in a child with acquired immune deficiency syndrome during rapid immune reconstitution. J Child Neurol 17: 457-60.

9. Kyle K, Maher M, Achhra AC, Venna N (2023) Contrasting cases of HIV vasculopathy associated fusiform aneurysms. Neurohospitalist 13: 69-3.

10. Pagiola IC, Paiva ALC, De Aguiar GB, Penalva De Oliveira AC, et al. (2016) Cerebral aneurysms associated with human immunodeficiency virus in adults: Literature review and new perspectives. Rev Assoc Med Bras 62: 85-9.