INDONESIAN HEALTH JOURNAL
https://ihj.rivierapublishing.id/index.php/rp
Volume 5 Number 2, June 2026
P-ISSN: 2963-3044
E-ISSN: 2963-3036

Dizziness and Cognitive Impairment in Parkinson's Disease Among HIV Patients: Case Report

Lathifatul Fikriyah*, Agnes Triana Basja, Safitri Indah Mashitah
Universitas Airlangga Academic Hospital, Surabaya, Indonesia
Email: lathifatul.fikriyah@fk.unair.ac.id*

Keywords:
HIV/ AIDS; dementia; parkinson; HIV-associated dementia

Abstract

Parkinsonism affects approximately 5% of individuals with HIV and represents the most common movement disorder in this population. It often coexists with HIV-associated neurocognitive disorder (HAND), opportunistic infections, or neuroleptic use. Accurate diagnosis of HIV-associated dementia (HAD) is crucial, as it is an AIDS-defining condition that may be reversible with antiretroviral therapy (ART). This case report aims to describe a patient with HIV who presented with dizziness, progressive cognitive decline, and parkinsonian features, highlighting the diagnostic challenges and the role of ART in management. A 41-year-old male with known HIV infection presented with progressive memory loss, dizziness, postural instability, bradykinesia, and intention tremors over several months. Neurological examination revealed cognitive impairment (MMSE 22/30) and parkinsonism (UPDRS-III 76/108). Brain MRI showed cerebral atrophy, white matter changes, and an abnormal "swallow tail" sign bilaterally in the substantia nigra, suggestive of Parkinson's disease. Laboratory tests demonstrated adequate systemic viral suppression (CD4 302 cells/μL). The patient was diagnosed with HIV-associated dementia and Parkinson's disease. His ART regimen was modified to enhance CNS penetration (zidovudine, lamivudine, dolutegravir). At two-month follow-up, he showed significant improvement in cognitive function and reduced dizziness, enabling return to work and daily activities. HIV-associated neurocognitive disorder and parkinsonism can occur even with well-controlled systemic HIV infection. A high index of suspicion, comprehensive neuropsychological and neuroimaging assessment, and individualized CNS-targeted ART are essential for optimal outcome.

INTRODUCTION

Dizziness is a frequently reported clinical complaint among older adults and individuals with neurodegenerative disorders, and it represents an important factor affecting functional mobility and quality of life (Joghataie, 2024; Lindell et al., 2024; McCaslin et al., 2025; Zhang et al., 2022). In patients with cognitive impairment or dementia, dizziness often correlates with vestibular dysfunction and postural instability, and it may coexist with deficits in attention and visuospatial cognition that exacerbate balance disturbances and fall risk. Evidence indicates that vestibular system dysfunction is linked to symptoms of dizziness in individuals with cognitive decline and postural instability, suggesting that sensory integration deficits contribute to the clinical presentation of dizziness in dementia-related conditions (Lee et al., 2020).

Between 4% and 15% of people with an HIV diagnosis have neurocognitive impairments as their main complaint. Nonspecific complaints including memory, concentration, attention, and motor skill deficiencies may be made by patients. Patients frequently have a range of mental or psychosocial issues that might impact cognitive function in addition to physical

comorbidities. These issues include mood disorders, post-traumatic stress disorder, and substance misuse or dependency. Neurological problems can also result from an increased incidence of malignancies, opportunistic infections, and ART medication side effects. In the later stages of the illness, patients may acquire dementia, or they may experience delirium as part of acute HIV syndrome (Singh et al., 2019; Kumar et al., 2018).

The range of progressively severe neurological and cognitive symptoms, formerly referred to as AIDS Dementia Complex (ADC), is now classified under the term HIV-associated neurocognitive disorder (HAND). In 2007, the United States National Institutes of Health established three distinct categories of HAND, which vary in severity: asymptomatic neurocognitive impairment, mild neurocognitive disorder, and HIV-associated dementia (HAD) (Zipeto et al., 2018). HIV infection can lead to a range of symptoms, including neurocognitive impairments (NCDs). Other minor symptoms may include apathy, sensory issues, and abnormalities in gait. Patients with HIV may experience cognitive problems known as HIV-associated neurocognitive dysfunction (HAND). Despite the effectiveness of combination antiretroviral therapy (cART) in reducing morbidity and mortality, many individuals living with HIV still experience HAND. Estimates suggest that up to 50% of these individuals may encounter some level of neurocognitive impairment during their illness (Dube et al., 2005).

HIV-Associated Neurocognitive Disorder (HAND) has a complex origin linked to immune system activation, chronic inflammation, neuronal damage, and the virus's direct effects on the central nervous system. Early in the infection, HIV can cross the blood-brain barrier and establish a reservoir within the central nervous system, making it challenging to eliminate the virus (Saylor et al., 2016; Valcour et al., 2012). Diffuse white matter abnormalities, particularly in the frontal and parietal lobes, are commonly observed on MRI scans of patients with HAND, and these findings correlate with the level of cognitive impairment (Hoare et al., 2015).

Movement disorders have been reported as a relatively rare manifestation in individuals with HIV infection. Two key studies have contributed significantly to the understanding of their prevalence among people living with HIV (PLH). De Mattos et al. reported a prevalence of 2.7% in a tertiary care hospital setting, whereas Mirsattari et al. found that approximately 50% of patients with AIDS exhibited parkinson features (Ferzana et al., 2023). Parkinsonism is a clinical syndrome of bradykinesia with either rigidity, tremor, or postural instability. The most common cause of Parkinsonism is PD. Other causes are drugs, infections, vascular events, or space-occupying lesions that affect the nigrostriatal pathway. PD is characterized by the progressive loss of dopaminergic (DA) neurons in the substantia nigra and pathological aggregates of alpha-Parkinsonism is a clinical syndrome characterized by bradykinesia, along with either rigidity, tremor, or postural instability. The most common cause of Parkinsonism is Parkinson's disease (PD), but other causes can include medications, infections, vascular events, or space-occupying lesions that impact the nigrostriatal pathway (Heaton et al., 2010).

This example highlights the importance of monitoring for symptoms of HIV-associated neurocognitive disorder (HAND) and Parkinson’s disease in patients with well-managed systemic HIV infection. Doctors should consider the possibility of CNS viral escape when making differential diagnoses. Furthermore, it is essential to promptly and appropriately adjust antiretroviral therapy (ART) regimens to address viral replication, specifically in the central

nervous system. Such adjustments can lead to significant clinical improvements, similar to the positive outcome experienced by this patient.

This case report aims to describe a 41-year-old male with well-controlled systemic HIV infection who developed progressive dizziness, cognitive decline, and parkinsonian features due to HIV-associated neurocognitive disorder and CNS viral escape, and to highlight the diagnostic challenges and the role of CNS-targeted antiretroviral therapy in achieving clinical improvement. This report provides clinical awareness for physicians managing HIV-positive patients who present with dizziness and cognitive or motor symptoms, emphasizing that HAND and parkinsonism should be considered even when systemic viral suppression appears adequate. It also offers practical guidance on modifying ART regimens to enhance CNS penetration, potentially reversing neurocognitive and motor decline, thereby improving patient quality of life and functional outcomes.

CASE REPORT

A 41-year-old man was undergoing antiretroviral therapy (ART) after being diagnosed with HIV in 2024. He began experiencing dizziness and cognitive decline a few months ago, and his condition worsened over time; he also experienced postural instability. The cognitive impairments made it challenging for him to perform everyday tasks and fulfill his job responsibilities. He reported symptoms such as slowed activities, severe memory loss, and significant difficulties with concentration. The patient reported a three-month history of non-vertiginous dizziness characterized by a persistent baseline sensation of lightheadedness and postural instability, accompanied by intermittent exacerbations lasting approximately 10–15 minutes. These episodes occurred several times per day and were predominantly precipitated by standing or abrupt changes in posture. There were no associated symptoms, including loss of consciousness, visual disturbances, palpitations, tinnitus, or hearing impairment.

The patient was cooperative and alert during the examination, although they displayed clear signs of cognitive impairment. They spoke slowly and used a limited vocabulary, exhibiting noticeable attention span and mathematical difficulties. A neurological assessment yielded a Mini-Mental State Examination (MMSE) score of 22 out of 30, indicating mild cognitive impairment. The Unified Parkinson’s Disease Rating Scale part III (UPDRS-III) score was 76 out of 108. Additionally, muscle strength scores in all four limbs were recorded at 4/5, suggesting minor widespread weakness. Gait evaluation demonstrated mild instability, which was more pronounced with eye closure. The Dix–Hallpike maneuver, conducted to evaluate a vestibular etiology of dizziness particularly benign paroxysmal positional vertigo was negative.

Laboratory tests showed a CD4 lymphocyte count of 302 cells/μL, indicating effective systemic viral suppression. Complete blood counts, liver function tests, renal function tests, and serum electrolyte levels were all within normal ranges.

A brain MRI revealed atrophy of the cerebrum, with an MTA score of 2, a GCA scale grade of I, and a Koedam scale grade of I. These findings may suggest the presence of presenile (early-onset) Alzheimer's disease. An abnormal “swallow tail” sign was observed bilaterally in the substantia nigra, raising suspicion of Parkinson's disease. There is a possibility of presenile Alzheimer's disease with Parkinsonism, which should be correlated with clinical findings. Furthermore, there is evidence of small vessel disease in the subcortical areas of the frontal and

parietal lobes on both the right and left sides (Fazekas grade 1). The brain MRA-TOF indicates aplasia of the right and left posterior communicating arteries (an embryonic variation). No stenosis or arteriovenous malformation was noted.

Based on the diagnosis of HIV-associated neurocognitive disorder (HAND) and central nervous system (CNS) viral escape, the patient's antiretroviral therapy (ART) regimen was modified to include zidovudine, lamivudine, and dolutegravir to enhance CNS penetration and more effectively suppress HIV replication within the CNS. The patient was initiated on pharmacological therapy consisting of betahistine administered at a dose of 24 mg twice daily, donepezil 10 mg once daily, and mecobalamin 500 µg twice daily. Betahistine was prescribed to alleviate dizziness-related symptoms, while donepezil was administered to address potential cognitive or central nervous system involvement. Mecobalamin was included as adjunctive therapy to support neurological function.

At a two-month follow-up, the patient showed significant clinical improvement and less frequent exacerbations of dizziness. His cognitive function had improved markedly, enabling him to resume both his employment and daily activities.

RESULTS AND DISCUSSION

We documented the case of a 41-year-old man whose HIV infection first manifested as rapidly advancing Parkinsonism. Following two months of ART therapy, our client showed improvements in both his cognitive performance and Parkinsonism. This case report raises the possibility that progressive dementia in older individuals living with HIV may result from HIV-associated neurocognitive disorder (HAND), Alzheimer's disease (AD), or a combination of both.

Cognitive decline in HIV-associated neurocognitive disorder (HAND) may be partially mediated by the accumulation of amyloid-β (Aβ) in the central nervous system (CNS) (Andras & Toborek, 2013). We propose that the individual described in this case may be experiencing mixed dementia, comprising features of both HAND and probable AD, although dementia due to either condition alone cannot be definitively excluded. Importantly, this case suggests that central nervous system (CNS) HIV infection does not preclude the deposition of amyloid-β (Aβ). Indeed, chronic HIV infection may act as a risk factor for AD through mechanisms such as persistent neuroinflammation, accelerated CNS aging, and diminished cognitive reserve, thereby constituting a potential "double-hit" to cognitive function.

The exact mechanism underlying the vulnerability of dopaminergic neurons to HIV infection remains unclear. In vitro studies have demonstrated that HIV can enter the brain shortly after the initial infection, when macrophages harboring the retrovirus cross the blood–brain barrier (Wang et al., 2004). Direct HIV infection has been demonstrated in macrophages and microglia, but not in neurons. Consequently, the neurotoxicity associated with HIV is believed to result primarily from the indirect effects of infected microglia. One proposed mechanism involves the HIV envelope glycoprotein 120 (gp120), which can bind to microglial cells. The subsequent release of proinflammatory cytokines by activated microglia may lead to neurotoxic effects on neighboring dopaminergic neurons (Reyes et al., 1991; Price et al., 1988).

Human immunodeficiency virus (HIV) infection is associated with a spectrum of neurological complications, ranging from HIV-associated neurocognitive disorders (HAND) to movement disorders, including Parkinsonism. Although dizziness per se has been less

frequently studied in HIV populations, HIV-related neurocognitive dysfunction is well documented and entails cognitive decline, gait abnormalities, and balance disturbances that can contribute to dizziness and increased fall risk (Amod et al., 2023). Movement disorders such as Parkinsonism can occur in people living with HIV. They may be related to HIV-induced neuronal injury or immune-mediated mechanisms affecting dopaminergic pathways, further complicating the presentation of balance and gait symptoms (Thompson, 2024).

Together, dizziness in dementia, Parkinson's disease, and HIV-associated neurocognitive and movement disorders reflect overlapping mechanisms of cognitive decline, autonomic dysfunction, and sensory integration disturbance. Improved recognition and comprehensive evaluation of dizziness in these populations are critical for optimizing management strategies, preventing falls, and enhancing overall functional outcomes (Karaca et al., 2022).

The patient's presentation of cognitive impairment, characterized by psychomotor slowing, memory loss, and generalized weakness, is consistent with a diagnosis of HIV-associated neurocognitive disorder (HAND). Brain MRI findings revealed atrophy of the cerebrum, with an MTA score of 2, a GCA scale grade of I, and a Koedam scale grade of I. These findings may support this diagnosis. White matter abnormalities are well-documented in HAND and frequently correlate with the degree of cognitive dysfunction, as observed in this case (Ragin et al., 2015).

The diagnosis of HAND accompanied by CNS viral escape necessitated a modification of the patient's antiretroviral therapy (ART) regimen. A combination of zidovudine, lamivudine, and dolutegravir was selected due to its improved central nervous system (CNS) penetration and efficacy in suppressing HIV replication within the CNS. Zidovudine and lamivudine, both nucleoside reverse transcriptase inhibitors (NRTIs), are recognized for their capacity to cross the blood–brain barrier. Dolutegravir, an integrase strand transfer inhibitor (INSTI), possesses potent antiretroviral activity and further contributes to CNS viral suppression (Rawson et al., 2018). This individualized therapeutic approach led to significant clinical improvement, with the patient exhibiting marked cognitive recovery and successfully returning to work within two months of the regimen modification.

CONCLUSION

This case highlights the complexities associated with the management of HIV-associated neurocognitive disorder (HAND) and people living with HIV (PLH), particularly in the context of central nervous system (CNS) viral escape. Despite effective systemic viral suppression with antiretroviral therapy (ART), the patient developed significant cognitive impairment due to localized HIV replication within the CNS. The marked clinical improvement observed following the adjustment of the ART regimen to include agents with enhanced CNS penetration underscores the critical importance of individualized, CNS-targeted treatment strategies in the management of HAND. Based on this case, clinicians evaluating HIV-positive patients with dizziness, cognitive decline, or motor symptoms should consider HAND and parkinsonism even when systemic viral suppression is adequate, perform comprehensive neuropsychological testing and brain MRI (including white matter and substantia nigra assessment), evaluate for CNS viral escape, and adjust ART to a regimen with higher CNS penetration effectiveness (e.g., zidovudine, lamivudine, and dolutegravir) while adopting a multidisciplinary approach. Future prospective studies are needed to establish standardized diagnostic criteria for HAND

with parkinsonism, assess long-term efficacy of CNS-targeted ART, and explore the mechanisms linking HIV neuroinflammation to dopaminergic injury.

REFERENCES

Amod, F., Holla, V. V., & Ojha, R. (2023). A review of movement disorders in persons living with HIV. *Parkinsonism & Related Disorders*, *114*, 105774. https://doi.org/10.1016/j.parkreldis.2023.105774

Andras, I. E., & Toborek, M. (2013). Amyloid beta accumulation in HIV-1-infected brain: The role of the blood brain barrier. *IUBMB Life*, *65*(1), 43–49.

Dube, B., Benton, T., Dean, G., et al. (2005). Neuropsychiatric manifestations of HIV infection and AIDS. *Journal of Psychiatry & Neuroscience*, *30*(4), 237–246.

Ferzana, A., & Vikram, V. (2023). A review of movement disorders in persons living with HIV. *Parkinsonism and Related Disorders*, *114*, 105774. https://doi.org/10.1016/j.parkreldis.2023.105774

Heaton, R. K., Clifford, D. B., Franklin, D. R., Jr., et al. (2010). HIV-associated neurocognitive disorders persist in the era of potent antiretroviral therapy: CHARTER study. *Neurology*, *75*(23), 2087–2096. https://doi.org/10.1212/WNL.0b013e318200d727

Hoare, J., Fouche, J. P., Spottiswoode, B., et al. (2015). White-matter damage in HIV-infected children with a history of encephalopathy. *AIDS Research and Therapy*, *2*, 461–470.

Joghataie, G. (2024). *Falls in older adults and patients with neurodegenerative disease: gaps in clinical practice and associations with cognitive and neuropsychiatric symptoms*. University of Toronto (Canada).

Karaca, S., Kalcioglu, M. T., Sargin, F., et al. (2022). Evaluation of effects of the human immunodeficiency virus on the vestibular system by using video head impulse test. *Eurasian Journal of Medicine*, *54*(2), 138–144.

Kumar, S., Maurya, V. K., Dandu, H. R., Bhatt, M. L., & Saxena, S. K. (2018). Global perspective of novel therapeutic strategies for the management of NeuroAIDS. *Biomolecular Concepts*, *9*(1), 33–42.

Lee, H. W., Lim, Y. H., & Kim, S.-H. (2020). Dizziness in patients with cognitive impairment. *Journal of Vestibular Research*, *30*(1), 17–23.

Lindell, E., Odhagen, E., & Tuomi, L. (2024). Living with dizziness impacts health-related quality of life among older adults. *Laryngoscope Investigative Otolaryngology*, *9*(1), e1194.

McCaslin, D. L., Berri, B., Miner, J. A., Graves, C. M., Gialloreti, K. L., Fansher, M., Kerber, K. A., Adams, M. E., & Carlozzi, N. E. (2025). Dizziness and its effect on health-related quality of life (DRQOL). *Journal of Vestibular Research*, 09574271251392050.

Price, R. W., Brew, B., Sidtis, J., Rosenblum, M., Scheck, A. C., & Cleary, P. (1988). The brain in AIDS: Central nervous system HIV-1 infection and AIDS dementia complex. *Science*, *239*(4840), 586–592.

Ragin, A. B., Wu, Y., Gao, Y., et al. (2015). Brain alterations within the first 100 days of HIV infection. *Annals of Clinical and Translational Neurology*, *2*(1), 12–21. https://doi.org/10.1002/acn3.136

Rawson, T., Muir, D., Mackie, N. E., et al. (2018). Central nervous system escape of HIV. *Current HIV/AIDS Reports*, *15*(2), 147–155.

Reyes, M. G., Faraldi, F., Senseng, C. S., Flowers, C., & Fariello, R. (1991). Nigral degeneration in acquired immune deficiency syndrome (AIDS). *Acta Neuropathologica*, *82*(1), 39–44.

Saylor, D., Dickens, A. M., Sacktor, N., et al. (2016). HIV-associated neurocognitive disorder — pathogenesis and prospects for treatment. *Nature Reviews Neurology*, *12*(4), 234–248. https://doi.org/10.1038/nrneurol.2016.27

Singh, H., Nain, S., Krishnaraj, A., Lata, S., & Dhole, T. N. (2019). Genetic variation of matrix metalloproteinase enzyme in HIV-associated neurocognitive disorder. *Gene*, *698*, 41–49.

Thompson, L. J. P. (2024). HIV-associated neurocognitive disorder: Mechanisms and challenges. *International Journal of Molecular Sciences*, *25*(9), 4697.

Valcour, V., Chalermchai, T., Sailasuta, N., et al. (2012). Central nervous system viral invasion and inflammation during acute HIV infection. *Journal of Infectious Diseases*, *206*(2), 275–282. https://doi.org/10.1093/infdis/jis326

Wang, G. J., Chang, L., Volkow, N. D., et al. (2004). Decreased brain dopaminergic transporters in HIV-associated dementia patients. *Brain*, *127*(11), 2452–2458.

Zhang, L., Jiang, W., Tang, L., Liu, H., & Li, F. (2022). Older patients with persistent postural-perceptual dizziness exhibit fewer emotional disorders and lower vertigo scores. *Scientific Reports*, *12*(1), 11908.

Zipeto, D., Serena, M., Mutascio, S., Parolini, F., Diani, E., Guizzardi, E., Muraro, V., Lattuada, E., Rizzardo, S., Malena, M., Lanzafame, M., Malerba, G., Romanelli, M. G., Tamburin, S., & Gibellini, D. (2018). HIV-1-associated neurocognitive disorders: Is HLA-C binding stability to β2-microglobulin a missing piece of the pathogenetic puzzle? *Frontiers in Neurology*, *9*, 791.