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PARKINSON'S DISEASE IN PATIENTS WITH HIV INFECTION

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Abstract
This article presents a literature review of cognitive impairment in Parkinson's disease in HIV-infected patients. Pathogenetic intersections between HIV-associated neurocognitive disorders (HAND) and Parkinsonism are analyzed. Neurodegeneration, with an emphasis on the role of the dopaminergic system, chronic neuroinflammation, and basal ganglia damage. This article describes the clinical and neuropsychological features of cognitive deficits associated with Parkinson's disease and HIV infection, along with data from modern neuroimaging and biomarker studies, as well as the impact of antiretroviral therapy and dopaminergic treatment on cognitive prognosis. The need for early cognitive screening and a multidisciplinary approach to managing this patient population is emphasized.

Keywords: Parkinson's disease; HIV infection; HIV-associated neurocognitive disorder (HAND); cognitive impairment; parkinsonism; dopaminergic system; neuroinflammation; neuroimaging; biomarkers.

Introduction

Cognitive impairment in Parkinson's disease in HIV-infected patients represents a complex, largely overlapping field of two neurodegenerative and neuroinflammatory processes. On the one hand, HIV-associated neurocognitive disorders (HIV-AND) neurocognitive Neurodegenerative disorders (HAND) remain one of the leading causes of cognitive decline in people living with HIV, despite the widespread adoption of combination antiretroviral therapy (ART). On the other hand, the aging of the HIV-positive population is leading to a natural increase in the prevalence of "classic" neurodegenerative diseases, including Parkinson's disease (PD). The intersection of these two trajectories—chronic HIV infection and the Parkinsonian process—creates a unique clinical and pathogenetic context for cognitive impairment, which has attracted increasing attention from researchers in recent years.

Even in the pre-war ART era, it was shown that HIV-related dementia and HIV-related encephalopathy are often accompanied by motor disorders, including bradykinesia, gait disturbances, postural instability, and other symptoms reminiscent of late stages of PD. Studies of the role of the basal ganglia and dopaminergic systems in HIV-related dementia have demonstrated significant involvement of the nigrostriatal system. system, hypodopaminergic state, and the presence of postural-kinetic disturbances similar to those seen in Parkinsonism. With the introduction of ART, the spectrum has changed: severe forms of HIV-associated dementia have become less common, but new clinical situations have emerged—either the development of

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parkinsonism as a manifestation of HIV-associated CNS damage or the manifestation of true Parkinson's disease in the setting of chronic HIV infection.

Epidemiological data on the incidence of parkinsonism and PD in HIV-positive patients remain fragmented, largely due to the difficulty of differentiating between various forms of movement disorders. Several reviews and clinical series indicate that movement syndromes (including parkinsonism) occur in approximately 5% of patients living with HIV, with the proportion of true PD among them being small. The wide range of causes of parkinsonism—from drug-induced extrapyramidal disorders and opportunistic infections to HIV-associated basal ganglia damage—makes it difficult to distinguish "pure" PD. A systematic review of movement disorders in HIV-positive patients (Amod et al., 2023) showed that parkinsonism can manifest during seroconversion, in the late stages of HIV infection, with drug toxicity, or as coinciding PD in elderly patients.

Chronic HIV infection itself is associated with a subcortical cognitive profile: slower processing speed, impaired attention, working memory, executive functions, and psychomotor speed. These domains largely overlap with those affected in Parkinson's disease, particularly in the early stages of Parkinsonian cognitive dysfunction (Parkinson's disease mild cognitive PD-MCI (HAND-mediated cognitive impairment, PD-MCI). PD is characterized by damage to frontostriatal and mesocortical circuits, leading to deficits in executive functions, attention, planning, and cognitive flexibility. As PD progresses, dementia develops with more pronounced impairments in memory, visuospatial abilities, and speech. Thus, there is significant overlap between HAND and cognitive impairment in PD at the cognitive profile level, complicating clinical differentiation in patients with a combination of these conditions.

The dopaminergic system is the pathogenetically central link linking HIV infection, HAND, and parkinsonian disorders . Early studies demonstrated that HIV-related dementia is accompanied by structural and functional changes in the striatum and other parts of the basal ganglia, as well as dysfunction of dopaminergic transmission. Modern neuroimaging studies using PET and SPECT confirm decreased activity of dopamine transporters and postsynaptic receptors in HIV-positive patients with cognitive impairment, even in the absence of clinically evident parkinsonism. Moreover, remodeling dopaminergic system is considered an important link in the formation of apathy, depression and executive deficits in HAND.

Interesting data show that chronic HIV infection and long-term ART are associated with subclinical motor disorders such as bradykinesia , detected using quantitative assessment methods such as quantitative digitography . A study by Prabhakar et al . (2020) showed that HIV patients aging with chronic infection exhibit subtle subcortical motor deficits resembling mild-moderate PD in their characteristics and correlating with cognitive decline. These data support the hypothesis of a partial overlap of pathogenic mechanisms—primarily, basal ganglia damage and neuroinflammation —between chronic HIV infection and early stages of Parkinsonism. neurodegeneration .

Clinically, parkinsonism in HIV-positive patients can manifest in at least three main patterns, each with a different association with cognitive impairment. The first pattern is HIV-associated parkinsonism associated with severe HIV encephalopathy or HIV dementia, often in patients not receiving ART. In such cases, motor symptoms (bradykinesia , rigidity, gait disturbances) are combined with subacute progressive subcortical cognitive decline, severe apathy, psychomotor retardation, and emotional and personality changes. Symmetrical, rapidly progressive parkinsonism

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is often observed, poorly responsive to standard dopaminergic therapy, but partially or completely regressing with effective ART, indicating a functional inflammatory nature to the disorder.

The second variant is coinciding Parkinson's disease in a patient with chronic, well-controlled HIV infection. In such cases, the clinical picture of PD (asymmetric resting tremor or rigid-bradykinetic syndrome, gradual progression) is largely reminiscent of idiopathic PD in HIV-negative patients. A series of observations by Moulignier et al . (2015) showed that HIV-associated PD is similar to idiopathic PD in its clinical and therapeutic characteristics: patients respond well to dopaminergic therapy, and the outcomes of surgical methods (deep stimulation of the subthalamic nucleus) are comparable to those in HIV-negative patients. Interestingly, some studies indicate a slower progression to severe stages of PD in HIV-positive patients, which is associated with possible functional adaptation of the dopaminergic system under the influence of chronic HIV infection.

The third variant is mixed and drug-induced parkinsonian syndromes, including neuroleptic parkinsonism, the toxic effects of certain antiretroviral drugs, and secondary parkinsonism secondary to opportunistic CNS lesions (toxoplasmosis, cryptococcal meningoencephalitis, progressive multifocal leukoencephalopathy). In these cases, the cognitive profile is variable and is determined by a combination of HAND, direct brain tissue damage from the infectious agent, medication effects, and comorbid psychiatric disorders.

Cognitive impairment in Parkinson's disease in HIV-positive patients is the result of the overlap, and sometimes synergy, of three groups of factors : Parkinsonian neurodegeneration , HIV-associated neuroinflammation with a subcortical -frontal HAND profile, as well as the influence of traditional vascular and metabolic risk factors, which are more common in aging patients with HIV.

In cases of coinciding PD and HIV, one might expect a more pronounced and earlier impairment of executive functions, attention, and psychomotor speed, given that these domains are affected in both HAND and parkinsonian cognitive dysfunction. The presence of pre-existing HAND may reduce the "reserve" of frontostriatal circuits, making the brain more vulnerable to additional dopaminergic stimulation. neurodegeneration .

Modern neuroimaging studies support the concept of complex damage to frontostriatal networks in HIV-positive patients with parkinsonian symptoms and cognitive impairment. Decreased gray matter volume in the frontal lobes, basal ganglia, and hippocampus, diffuse white matter changes, and impaired functional connectivity in the default mode and frontoparietal networks have been noted . In true PD against the background of HIV, structural changes in the nigrostriatal system (decreased accumulation of dopaminergic radioligands in the striatum) are combined with features of diffuse subcortical damage characteristic of HAND, which may explain the more complex cognitive phenotype compared to “pure” PD.

In terms of biomarkers, neurofilament light chains (NfL) as a marker of axonal injury and various indicators of monocyte- microglial activation appear to be the most promising for assessing cognitive impairment in patients with PD and HIV. Elevated NfL levels in plasma and cerebrospinal fluid have been described in both HAND and PD and other neurodegenerative processes; an additive effect can be expected when combined. Data on specific differences in NfL profiles and other markers (GFAP, cytokines, chemokines) in patients with coinciding PD and HIV are still limited, but current reviews highlight the potential of a multimodal approach including neuroimaging and biomarkers for stratifying the risk of cognitive decline.

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Differential diagnosis presents a separate challenge: in an HIV-positive patient with Parkinsonism and cognitive decline, it is necessary to distinguish between HIV-associated subcortical dementia, PD with dementia, vascular cognitive dysfunction, and mixed conditions. Classically, HAND is characterized by more pronounced slowness, attention deficits, and executive functions with relative preservation of episodic memory and visuospatial skills in the initial stages, whereas in dementia associated with PD and comorbid pathology such as Alzheimer's disease, memory and visuospatial impairments may become more prominent. The presence of marked asymmetry in motor symptoms, a good response to levodopa, the typical evolution of the motor syndrome for PD, and characteristic changes on dopaminergic SPECT/PET support the diagnosis of PD but does not exclude concomitant HAND.

Current clinical observations show that HIV infection does not impair the response to dopaminergic therapy in Parkinson's disease and does not make PD more "malignant" in terms of outcomes. On the contrary, some series have noted a slower progression to severe stages in HIV-positive patients with PD with comparable levodopa doses. However, the cognitive prognosis may be less favorable given the presence of underlying HAND and the influence of ART, psychotropic, and other medications. The risk of drug interactions (including between antiretrovirals and dopamine agonists, MAO-B inhibitors, etc.) requires careful selection of treatment regimens.

An important area of research is assessing the impact of ART on the risk and severity of parkinsonism and cognitive impairment. Certain medications (e.g., efavirenz) are associated with neurological and psychiatric side effects, including sleep and mood disturbances, cognitive complaints, and, in rare cases, movement disorders. At the same time, effective suppression of viral replication and a reduction in chronic neuroinflammation with modern ART regimens (especially those using integrase inhibitors) help reduce the severity of HAND and likely decrease the risk of HIV-associated parkinsonism. Currently, there is no convincing data on the direct impact of ART on the risk of true PD, but the hypothesis of modification of the " neuroinflammatory background," which may be a risk factor for neurodegeneration , is being considered .

In terms of functional outcomes, the combination of cognitive impairments associated with PD and HIV has significant consequences for activities of daily living, treatment adherence, and quality of life. Systematic reviews show that HAND significantly impairs the ability to plan, perform complex daily tasks, manage medication regimens, and manage finances. Similarly, cognitive impairment in PD (especially when accompanied by depression and apathy) significantly reduces functioning and increases the burden of care. When these conditions are combined, a cumulative effect can be expected, necessitating early identification of cognitive deficits, the involvement of a multidisciplinary team (neurologist, infectious disease specialist, neuropsychologist, psychiatrist, rehabilitation specialist), and the development of individualized support programs.

Current research emphasizes the importance of early and regular cognitive screening in all HIV-positive patients with motor symptoms resembling Parkinson's disease, as well as in patients with established Parkinson's disease and HIV infection. The use of validated brief scales (MoCA , IHDS, specialized Parkinsonian cognitive scales) in combination with in-depth neuropsychological testing allows for the identification of mild and moderate forms of cognitive dysfunction even before the development of overt dementia. In the future, multidomain models integrating cognitive data,

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neuroimaging, and biomarkers may enable more accurate prognoses and personalized therapy for both HAND and cognitive impairment associated with Parkinson's disease in HIV-positive patients.

Conclusions

In summary, cognitive impairment in Parkinson's disease in HIV-infected patients is a multicomponent phenomenon, based on the interaction of neurodegenerative, neuroinflammatory , and vascular-metabolic processes. HIV-associated damage to the basal ganglia and dopaminergic pathways creates a "vulnerable substrate" for parkinsonism. Neurodegeneration and worsens the subcortical cognitive profile, while natural aging and comorbidity increase the risk of classic PD and vascular cognitive dysfunction. Current studies, including systematic reviews of movement disorders in HIV, descriptions of patient series with HIV-associated PD, neuroimaging and biomarker studies, indicate a partial clinical and pathogenetic commonality between HAND and cognitive impairment in PD, but also emphasize the need for larger prospective studies and the development of standardized diagnostic criteria for this group of patients. In practical terms, early cognitive screening, optimization of ART, careful selection of dopaminergic therapy, and a multidisciplinary approach remain key tools in the management of cognitive and motor impairment in patients with HIV and Parkinson's disease.

References

1. Sarvinoz, T., & Muzaffar, Z. (2022). Rehabilitation for childhood cerebral palsy. *Uzbek Scholar Journal*, *6*, 97-101.
2. Nabievna, M. Y., & Muzaffar, Z. (2023). Modern View on the Pathogenesis of Hiv Encephalopathy.
3. Sarvinoz, T., & Muzaffar, Z. (2022). Rehabilitation aspects of water therapy in modern medicine. *Uzbek Scholar Journal*, *6*, 102-106.
4. Nabievna, M. Y., & Muzaffar, Z. (2022). Literatural review of the relevance of the problem of neurosaids. *Modern Journal of Social Sciences and Humanities*, *4*, 558-561.
5. Mukhammadjonov, O. Zokirov M. (2022). Cognitive impairments in patients with HIV-associated encephalopathy. *Central Asian Journal of Medical and Natural Science*, *3*(2), 401-405.
6. Зокиров, М.М., Мухаммаджонов, О. (2022). Когнитивные нарушения у пациентов с болезнью Паркинсона и оптимизация ее лечения.
7. Зокиров, М., & Туланбоева, С. (2022). Когнитивные нарушений у пациентов с ВИЧ–ассоциированной энцефалопатией. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 68-73.
8. Зокиров, М., & Мухаммаджонов, О. (2022). Особенности развития тревожных и депрессивных расстройств при заболеваниях, сопровождающихся хроническим болевым синдромом. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 841-844.
9. Muzaffar, Z., & Okilbeck, M. (2022). Dementia and arterial hypertension. *Modern Journal of Social Sciences and Humanities*, *4*, 19-23.
10. Muzaffar, Z. (2022). HIV Encephalopathy and its Pathogenetic Aspects. *European Multidisciplinary Journal of Modern Science*, *4*, 843-846.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

11. Muzaffar, Z. (2022). Literature reviews on nervous system damage during hiv infection. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, *2*(9), 141-147.
12. Зокиров, М., & Мухаммаджонов, О. (2022). Вич энцефалопатия и его патогенетические аспекты. *Barqarorlik va yetakchi tadqiqotlar onlayn ilmiy jurnali*, 855-858.
13. Зокиров, М. М., Рустамова, И. К., Касимова, С. А., & Кучкарова, О. Б. (2019). Жарохатдан кейинги талвасада кечки нейровизуализацион ўзгаришлар. In *Современная медицина: новые подходы и актуальные исследования* (pp. 56-60).
14. Нессипхан, Б., Шамих Аль-Саеди, Х.Ф., Джассим Аль-сарай, М., Хамид Джасим, Х., Саттар, Р., Али Ахмед, Б., ... и Музаффар, З. (2023). Применение наночастиц оксида железа в качестве усилителей контрастности МРТ: исследование на кроликах. *Журнал наноструктур* , *13* (3), 769-776.
15. Zokiriv, M. (2021). Correction of cognitive impairments in patients with HIV-associated encephalopathy. *J. Theor. Appl. Sci*, *7*, 62-66.
16. Muzaffar, Z. (2022). Psychological State in Patients with HIV Infection. *Amaliy va tibbiyot fanlari ilmiy jurnali*, *1*(6), 52-56.
17. Zokirov, M. M., & Madjidova, Y. N. (2020). Correction Of Cognitive Disorder In Patients With HIV-Associated Encephalopathy. *The American Journal of Medical Sciences and Pharmaceutical Research*, *2*(07), 117-122.
18. Зокиров, М., & Мадмаров, Д. (2022). Корреляция ЭЭГ картины головного мозга и когнтитивного статуса у пациентов с эпилепсией. *Theoretical aspects in the formation of pedagogical sciences*, *1*(5), 227-230.
19. Жахонкуловна С.М., Камоловна Г.Б., Зокиров М., Таджимуратовна Б.У., Юмашев А., Шичьях Р., ..., Ишанкулов А. (2025). Электрохимические биосенсоры для раннего выявления болезни Альцгеймера. *Клиника Химика Акта* , 120278.
20. Muzaffar, Z., & Zarifa, Z. (2023). Yosh bemorlarda gemorragik insultning klinik holatlari. *Psixologiya va sotsiologiya ilmiy jurnali*, *1*(1), 29-31.
21. Zokirov, M. (2023). Features of cognitive impairment in patients with HIV encephalopathy. In *Academic International Conference on Multi-Disciplinary Studies and Education* (Vol. 1, No. 9, pp. 34-36).
22. Muhammadjonov, o., & Zokirov, m. 2-toifa qandli diabet bilan og'rigan bemorlarda yurak-qon tomir kasalliklarining xavf omillarining tarqalishi. *Студенческий вестник Учредители: Общество с ограниченной ответственностью" Интернаука"*, 53-54.
23. Zokirov, M. M. (2025). Characteristics of post-stroke depression in recurrent ischemic stroke. *Журнал гуманитарных и естественных наук*, (24), 23-27.
24. Muzaffar, Z., & Shirin, D. (2024). Damage to the nervous system due to HIV. *Eurasian Journal of Technology and Innovation*, *2*(7), 39-47.
25. Madjidova, Y., & Zokirov, M. (2024). Cognitive disorders in HIV encephalopathy. *Science and innovation*, *3*(D5), 375-381.

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