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CASE PRESENTATION

Progressive multifocal leukoencephalopathy by JC Polyomavirus as an initial manifestation of HIV

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ABSTRACT

Introduction: progressive multifocal leukoencephalopathy is a demyelinating disease of the central nervous system caused by the reactivation of the JC polyomavirus, which typically occurs in contexts of severe immunosuppression. Its clinical presentation is often devastating and evolves rapidly towards disability and death.
Objective: to present a clinical case of fulminant neurological onset associated with advanced HIV infection, illustrating the importance of early diagnosis and timely access to comprehensive treatment.
Case presentation: we present the case of a 36-year-old male with no medical history, who consulted for progressive weakness in the right hemibody, dysarthria, and behavioral changes. The neurological examination confirmed right hemiparesis and decreased level of attention. Serology revealed HIV infection with a viral load >750,000 copies/mL and CD4 T-cells at 18 cells/mm³. Brain MRI showed bilateral parieto-occipital white matter lesions, without enhancement or mass effect, compatible with PML. PCR in cerebrospinal fluid was positive for JC polyomavirus, ruling out other opportunistic infections. Antiretroviral treatment was initiated under surveillance for IRIS and neurorehabilitation support. The patient presented progressive neurological deterioration and died three weeks after admission.
Conclusions: this case illustrates a form of fulminant neurological onset of advanced HIV, with irreversible evolution. The fatal outcome underscores the urgency of public health strategies aimed at early detection and reduction of severe immunosuppression in patients with HIV.

Keywords: Hiv Infections; Leukoencephalopathy, Progressive Multifocal; Jc Virus.

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INTRODUCTION

Progressive multifocal leukoencephalopathy (PML) is a serious demyelinating disease of the central nervous system caused by the reactivation of the JC polyomavirus, a neurotropic virus that infects more than 50 % of the general population in a latent form.[1] In immunocompetent individuals, the infection remains asymptomatic; however, in contexts of profound immunosuppression—such as in patients with HIV/AIDS, hematological neoplasms, or under immunomodulatory treatment—the virus can reactivate and cause progressive, irreversible, and potentially fatal brain damage.[2,3]

In the context of HIV, PML represents a late-onset opportunistic complication, associated with CD4 lymphocyte counts below 100 cells/mm³.[4] Before the era of antiretroviral therapy (ART), it was estimated that up to 5 % of patients with AIDS developed PML.[5]

Despite advances in the knowledge of PML and its association with advanced immunosuppression, early diagnosis continues to be a challenge due to the variability of clinical and radiological presentation. In particular, cases where PML constitutes the initial manifestation of HIV are rarely reported in the region and pose a significant diagnostic challenge, given that neurological symptoms can be confused with other entities.[6,7] This report describes a case of fulminant neurological onset due to PML in a young patient with no medical history, whose rapid and irreversible evolution underscores the need to strengthen screening and comprehensive care strategies in contexts of high HIV burden.

CASE PRESENTATION

A 36-year-old male patient with no relevant medical history or known chronic treatments. He reported no recent travel, occupational exposure to risk agents, or intravenous drug use. He denied the use of immunosuppressants, previous transfusions, or diagnosed sexually transmitted diseases. He presented to the emergency service with a subacute neurological condition of three weeks' evolution, starting with progressive weakness in the right hemibody, difficulty articulating words, episodes of irritability, and social isolation. In the last few days, temporospatial disorientation, slowness in verbal responses, and decreased interaction with the environment were added. No fever, headache, seizures, or systemic symptoms were reported.

On physical examination, right hemiparesis with brachial predominance, moderate dysarthria, decreased level of attention, and psychomotor slowness were noted. The patient was afebrile, with stable vital signs, without meningeal signs or alterations in cranial nerves. The rest of the clinical examination was normal.

Given the presence of right hemiparesis, dysarthria, and behavioral changes in a young patient with no history, differential diagnoses such as cerebrovascular accident, viral encephalitis, and expansive processes were initially considered.

Laboratory studies were requested, where HIV serology was positive, with a viral load greater than 750,000 copies/mL and CD4+ T-lymphocytes at 18 cells/mm³. The positive serology with very low CD4+ count guided the reasoning towards advanced immunosuppression, which increased the suspicion of opportunistic infections of the central nervous system. The blood count showed mild leukopenia and lymphopenia, without anemia or thrombocytopenia. Liver and kidney tests were normal.

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A computed tomography scan of the brain was performed without acute findings, which reduced the probability of a vascular event. Subsequently, a brain magnetic resonance imaging (MRI), performed on the second day of hospitalization, evidenced multifocal lesions in the parieto-occipital white matter, hypointense on T1 and hyperintense on T2/FLAIR, without contrast enhancement or mass effect. The absence of edema and contrast uptake was key to ruling out expansive processes or infections with an active inflammatory component. These findings were suggestive of PML (Figure 1).

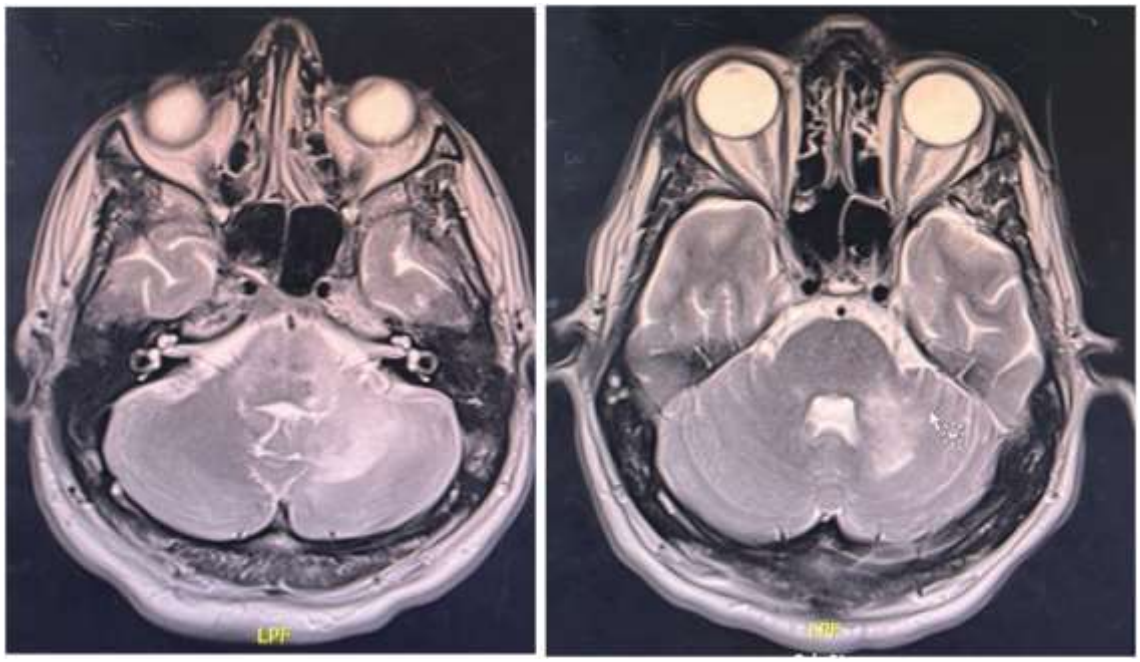

Fig. 1 Brain magnetic resonance imaging with lesions in the left parieto-occipital white matter.

To confirm the diagnosis and rule out alternative infections, a lumbar puncture was performed. The cerebrospinal fluid was clear, without pleocytosis or hypoglycorrhachia, findings that made bacterial or viral meningitis unlikely. PCR was positive for JC polyomavirus and negative for Cryptococcus, Toxoplasma, CMV, and HSV. Brain biopsy was not performed due to the high risk and because the combination of clinical, imaging, and virological findings already offered sufficient diagnostic certainty.

With these results, the definitive diagnosis of PML as an initial manifestation of advanced HIV was established. Given the absence of specific antiviral therapies with proven efficacy for PML, it was decided to initiate antiretroviral therapy (ART) under strict surveillance, recognizing that immune reconstitution constitutes the main therapeutic strategy.

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During the evolution, the patient presented immune reconstitution inflammatory syndrome (IRIS), which was managed through corticosteroid therapy adjusted to the clinical response, along with symptomatic support measures and prevention of infectious complications. Neurorehabilitation support and multidisciplinary follow-up by neurology, infectiology, and internal medicine were also implemented, with the aim of optimizing functional recovery and documenting both the successes and adverse factors of the therapeutic approach.

During hospitalization, the patient presented progressive neurological deterioration, with decreased level of consciousness, loss of verbal response, intermittent postural rigidity, and episodes of disconnection from the environment. No convulsive crises or signs of intracranial hypertension were documented. Despite the interventions instituted, he evolved into deep coma and died three weeks after hospital admission. After death, an autopsy was offered for academic and diagnostic confirmation purposes; however, this was declined by the family.

DISCUSSION

Progressive multifocal leukoencephalopathy (PML) is a serious neurological condition characterized by extensive demyelination in the central nervous system, secondary to the reactivation of the JC polyomavirus.[2] This virus, of high prevalence in the general population, usually remains latent without causing symptoms; however, in situations of profound cellular immunosuppression—such as in HIV/AIDS—it can reactivate and cause progressive and irreversible neurological deterioration. Given the absence of specific antiviral treatments, the only available therapeutic strategy consists of restoring immunity through ART.[5]

In Latin America, the epidemiology of PML is underreported, due to low clinical suspicion, limited availability of neuroimaging studies, and scarce accessibility to PCR tests for JC polyomavirus in cerebrospinal fluid.[8] In Paraguay, there are no consolidated national records on the incidence of PML, but isolated cases have been reported in patients with advanced HIV, especially in reference hospitals.[9] The persistence of late HIV diagnoses, along with structural barriers in access to ART, contributes to the appearance of serious neurological complications such as PML.[10] The present case, occurring in a young patient with no medical history, evidences the need to strengthen screening, follow-up, and comprehensive care strategies in the region.

Regarding the clinical presentation of PML, it usually includes subacute focal neurological deficits, such as hemiparesis, dysarthria, cognitive alterations, and behavioral changes.[11] In this case, the patient presented progressive weakness in the right hemibody, difficulty articulating words, and disorientation, without fever or meningeal signs. This symptomatic triad, in the absence of systemic infectious signs, coincides with what is described in European and Latin American series as the initial pattern of PML in patients with late HIV diagnosis.[9,12,13]

Brain magnetic resonance imaging showed multifocal lesions in the bilateral parieto-occipital white matter, without mass effect or contrast enhancement, findings considered characteristic of PML.[7] Etiological confirmation was obtained through positive PCR for JC polyomavirus in cerebrospinal fluid, a test that is currently considered the diagnostic standard due to its high sensitivity and specificity.[14] The exclusion of other opportunistic infections—cryptococcosis, toxoplasmosis, cytomegalovirus, and herpes simplex—through serological and molecular studies was key to establishing the definitive diagnosis. In patients with HIV, the differential diagnosis must also include primary lymphoma of the central nervous system and viral encephalitis, entities that share clinical and radiological manifestations.[15]

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The diagnostic approach to PML requires precise integration of clinical, imaging, and virological data. In Paraguay, although there are no consolidated epidemiological records on the incidence of PML, isolated cases have been reported in reference hospitals, suggesting an underestimation of its real prevalence.[15] Low clinical suspicion, limited availability of neuroimaging studies, and restricted access to PCR tests for JC virus contribute to this underreporting. This context reinforces the importance of considering PML in the differential diagnosis of neurological conditions in immunosuppressed patients, especially in regions with high HIV burden and unequal access to diagnosis and treatment.

Although currently there is no specific antiviral treatment with proven efficacy for PML, experimental or adjuvant therapies—such as mefloquine, maraviroc, cytarabine, and other immunomodulatory agents—have been described with variable results and without consensus in international guidelines.[6,7,10,12] In this case, these options were not considered due to the limited evidence available, the absence of regulated access in our setting, and the patient's clinical status, which did not allow the safe implementation of experimental protocols.

The active management of immune reconstitution inflammatory syndrome (IRIS) was a crucial aspect. The patient presented manifestations compatible with IRIS after the initiation of ART, which motivated the use of corticosteroid therapy adjusted to clinical evolution, in accordance with recommendations for severe cases.[13] This approach sought to mitigate the excessive inflammatory response without compromising the efficacy of immune reconstitution.

Finally, it is relevant to analyze whether the initiation of ART could have accelerated neurological deterioration via IRIS. Although immune reconstitution constitutes the fundamental therapeutic strategy against PML, in some patients it can precipitate a disproportionate inflammatory response that aggravates neurological damage.[14] In this case, the rapid clinical progression suggests that IRIS contributed significantly to the adverse outcome, which underscores the need for close surveillance and individualized strategies at the start of ART in patients with advanced HIV and neurological compromise.

The adverse outcome in this case reflects not only the aggressiveness of PML and current therapeutic limitations, but also the importance of timely management of advanced HIV and IRIS. The experience underscores the need for close surveillance and individualized strategies when initiating ART in patients with neurological compromise. From this clinical analysis, it becomes inevitable to consider how inequalities in access to early diagnosis, support therapies, and specialized resources condition the evolution of patients. In contexts of greater vulnerability, these gaps translate into more unfavorable outcomes, which reinforces the urgency of equity policies in health and universal access to comprehensive care.

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Conflict of Interest
The authors declare that there is no conflict of interest.

Authorship Contribution
MAMA: conceptualization, methodology, investigation, writing – original draft, writing – review and editing.
VMCC: conceptualization, validation, investigation, supervision, writing – original draft, writing – review and editing.

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CGD: methodology, validation, investigation, writing – original draft, writing – review and editing.

Ethical Aspects
All ethical standards have been respected. Consent has been received from the patient's guardians (parents) for the publication of this work. The manuscript has not been submitted simultaneously to other journals for publication.

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