

A Case Series of Unusual Neurological Presentation in HIV Positive Patients and Their Outcome in Tertiary Care Teaching Institute**Pravin Soni¹, Niranjana Pathak², Manisha Thakur³**¹Professor & HOD, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune, India²Associate Professor, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune, India³Resident, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune, India

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Abstract

We report herein an interesting case series of admitted patients in YCM Hospital presenting with HIV infection. It includes 6 patients admitted from April 2024 to March 2025. Out of 6 patients 4 were male and 2 were female. People living with HIV infection are more prone for opportunistic CNS infections due to immunocompromised status. Early diagnosis, timely intervention can save patients from permanent neurological deficit and from death.

Keywords: HIV Infection, Neurological Manifestations.**DOI:** 10.25258/ijcpr.18.8.224

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Introduction

Acquired Immunodeficiency Syndrome (AIDS), the most advanced stage of Human Immunodeficiency Virus (HIV) infection, is characterized by profound immune suppression that predisposes individuals to a wide range of opportunistic infections (OIs). HIV primarily targets CD4⁺ T lymphocytes, leading to progressive immune deterioration. The risk and spectrum of OIs are closely correlated with declining CD4 cell counts, with more severe and life-threatening infections occurring at lower levels [1]. Opportunistic infections serve as key clinical indicators of immune compromise in HIV-positive individuals and often define the progression from HIV infection to AIDS. For instance, oropharyngeal candidiasis and tuberculosis may occur at CD4 counts below 500 cells/ μ L, while infections like *Pneumocystis jirovecii* pneumonia (PCP) and cryptococcal meningitis are more frequently observed when CD4 counts fall below 200 cells/ μ L [2,3]. Cytomegalovirus retinitis and disseminated *Mycobacterium avium* complex (MAC) infections typically present at CD4 counts under 50 cells/ μ L [4].

The incidence of OIs has declined significantly in regions with widespread access to combination antiretroviral therapy (ART), yet they remain a major cause of morbidity and mortality in individuals with delayed diagnosis, irregular treatment, or ART failure [5]. Understanding the relationship between CD4 count and susceptibility

to specific OIs is crucial for early diagnosis, prophylaxis, and appropriate therapeutic interventions

Opportunistic infections in HIV includes CNS tuberculosis, cryptococcal meningitis, HIV encephalitis, toxoplasmosis etc.

Criteria for inclusion of patients for the study:

Any case of altered sensorium admitted to the Department of Medicine in PGI Yashwantrao Chavan Memorial Hospital, PCMC, and Pune who is:

- Age >18 years.
- Patients living with HIV/AIDS

Criteria for exclusion of patients for the study

1. Traumatic etiology
2. Substance abuse disorder

Case Presentation

Case 1: Cryptococcal meningitis is life-threatening neurological infection, typically occurring in advanced HIV disease with CD4 counts below 100 cells/ μ L. It often presents with subacute meningoencephalitis characterized by headache, fever, and altered mental status. In this series, two patients presented with cryptococcal meningitis—one of whom succumbed despite antifungal therapy, while the other showed significant recovery following treatment. Standard management includes induction therapy with

amphotericin B and flucytosine, followed by consolidation and maintenance therapy with fluconazole. ART initiation is usually deferred by 4–6 weeks after starting antifungal treatment to reduce the risk of immune reconstitution inflammatory syndrome (IRIS) [7,8].

Case 2: Tuberculosis (TB) is one of the most prevalent OIs among people living with HIV (PLHIV), particularly in endemic regions. In the present series, a 65-year-old male on long-term ART with a past history of pulmonary TB developed tubercular meningitis confirmed through imaging and CSF analysis showing acid-fast bacilli (AFB) and positive for CBNAAT. This presentation aligns with the known predilection of TB to involve extrapulmonary sites in immunocompromised hosts, and early initiation of anti-Koch's therapy (AKT) led to clinical improvement [6].

Case 3: HIV-associated encephalitis is a neurological manifestation that may occur during advanced or even early stages of HIV infection. The patient's symptoms—seizures, memory loss, and behavioral changes like cognitive impairment. Seizures in HIV-positive individuals may result from direct viral effects, opportunistic infections, or metabolic abnormalities. [10] However, in this case, other CNS infections like herpes simplex virus (HSV), tuberculosis, toxoplasmosis, and cryptococcal meningitis were ruled out before initiating antiretroviral therapy (ART), strengthening the diagnosis of primary HIV encephalitis. The MRI findings of hyperintensities in the periventricular white matter are characteristic of HIV encephalopathy and reflect demyelination or gliosis secondary to viral infiltration of the CNS. [11] CSF analysis revealed elevated proteins without significant pleocytosis. Prompt initiation of

ART is the cornerstone of treatment for HIV encephalitis. Studies have shown that early ART improves cognitive outcomes and reduces viral replication in the central nervous system (CNS). [12]

Case 4: Cerebral toxoplasmosis is the most common cause of focal brain lesions in patients with AIDS and a CD4 count below 100 cells/ μ L. It is usually due to reactivation of latent *Toxoplasma gondii* infection. Diagnosis is often presumptive based on clinical presentation, imaging, and serology, with a positive response to therapy considered confirmatory [13]. MRI typically shows multiple ring-enhancing lesions, often in the basal ganglia or corticomedullary junction [14]. Empiric therapy is started in most cases, with brain biopsy reserved for non-responders or diagnostic uncertainty. Timely initiation of anti-toxoplasma therapy, corticosteroids (if there's significant edema), and delayed ART initiation are critical to avoid IRIS and optimize outcomes.

Case 5: Progressive multifocal leukoencephalopathy (PML) in an untreated HIV-positive patient, presenting with focal neurological deficits and cognitive impairment. MRI findings and positive JC virus PCR in CSF confirmed the diagnosis. Initiation of ART led to the development of immune reconstitution inflammatory syndrome (IRIS), a known complication due to immune recovery.

The IRIS was managed conservatively without corticosteroids, given the absence of severe symptoms. The patient's gradual improvement and discharge highlight the importance of early diagnosis and careful monitoring during ART initiation.

Results

Table 1: Distribution of Etiologies in neurological manifestation in HIV/AIDS (n=6)

Etiology	Number of cases (n)
Cryptococcal Meningitis	2
Tubercular Meningitis	1
Cerebral Toxoplasmosis	1
HIV Encephalopathy	1
Progressive Multifocal Leukoencephalopathy	1

Table 2: Clinical features across different etiologies

Clinical Feature	Cryptococcal Meningitis	Tubercular Meningitis	HIV Encephalitis	Cerebral Toxoplasmosis	Progressive Multifocal Leukoencephalopathy
Acute Onset (<72 hrs)	Yes	Yes	Yes	Yes	no
Headache	Yes	Yes	Yes	Yes	no
Fever	Yes	Yes	Yes	Yes	No
Altered Sensorium	Yes	Yes	Yes	Yes	No
Convulsions	Yes	No	No	Yes	No
Neck Rigidity	Yes	Yes	No	No	No
Focal neurological Deficit	No	No	No	No	Yes
CD4 count	100	168	260	65	45

Table 3: Treatment and Outcomes

Parameter	Cryptococcal Meningitis	Tubercular Meningitis	HIV Encephalitis	Cerebral Toxoplasmosis	Progressive Multifocal Leukoencephalopathy
Antiretroviral Therapy	No	Yes	Yes	No	yes
Antitubercular Therapy	No	Yes	No	No	No
Antifungal Drugs	Yes	No	no	No	No
Anticonvulsant	Yes	Yes	Yes	Yes	Yes
Ventilator Support Needed	Yes	No	No	Yes	No
Full recovery at discharge (%)	Death	Yes	Yes	Death	No
Mortality	1/2cases	0%	0%	100%	0%

Discussion

Cryptococcal Meningitis: Life-threatening fungal infection of the meninges caused primarily by *Cryptococcus neoformans* and less commonly by *Cryptococcus gattii*. It mainly affects immunocompromised individuals, especially those with HIV/AIDS, organ transplant recipients, or patients on long-term corticosteroids. Infection typically begins in the lungs after inhalation of spores and spreads hematogenously to the central nervous system. Symptoms include headache, fever, neck stiffness, photophobia, altered mental status, and raised intracranial pressure. Diagnosis is confirmed by cerebrospinal fluid analysis with India ink staining, cryptococcal antigen testing, and culture. Treatment involves amphotericin B plus flucytosine, followed by prolonged fluconazole therapy.

Tubercular Meningitis (TB meningitis): Severe form of central nervous system tuberculosis caused by *Mycobacterium tuberculosis*. It results from hematogenous spread of infection from a primary focus, often pulmonary tuberculosis, leading to inflammation of the meninges and formation of basal exudates. It is more common in children and immunocompromised individuals, especially those with HIV/AIDS. Symptoms develop gradually and include persistent fever, headache, vomiting, neck stiffness, altered consciousness, and cranial nerve palsies. Complications include hydrocephalus, stroke, and neurological deficits. Diagnosis is based on cerebrospinal fluid analysis, imaging, and microbiological tests. Treatment requires prolonged anti-tubercular therapy with adjunctive corticosteroids.

HIV Encephalitis: A neurological complication of advanced HIV/AIDS caused by direct infection of the central nervous system by Human immunodeficiency virus. The virus crosses the blood–brain barrier via infected macrophages, leading to chronic inflammation, microglial activation, and neuronal damage. It commonly

presents with progressive cognitive decline, memory impairment, behavioral changes, motor dysfunction, and slowed thinking, collectively termed HIV-associated neurocognitive disorder (HAND). Symptoms usually develop gradually in untreated or poorly controlled HIV infection. Diagnosis is clinical, supported by neuroimaging and exclusion of opportunistic infections. Management involves effective antiretroviral therapy, which can improve or stabilize neurological function and reduce further central nervous system damage.

Cerebral Toxoplasmosis: A serious opportunistic infection of the brain caused by reactivation of latent infection with *Toxoplasma gondii*, particularly in individuals with advanced HIV/AIDS. It commonly occurs when CD4 counts fall below 100 cells/ μ L. The parasite forms cysts in neural tissue, leading to focal inflammation and necrosis. Clinical features include headache, fever, confusion, seizures, focal neurological deficits, and altered consciousness. Neuroimaging typically shows multiple ring-enhancing lesions, especially in basal ganglia. Diagnosis is based on clinical presentation, imaging, and serology. Treatment includes pyrimethamine, sulfadiazine, and leucovorin, along with antiretroviral therapy and prophylaxis to prevent recurrence.

Progressive Multifocal Leukoencephalopathy (PML): A rare, often fatal demyelinating disease of the central nervous system caused by reactivation of the John Cunningham virus (JC virus) in immunocompromised individuals, especially those with HIV/AIDS or on immunosuppressive therapies. The virus infects and destroys oligodendrocytes, leading to multifocal areas of white matter demyelination. Symptoms progress over weeks to months and include weakness, visual disturbances, speech impairment, cognitive decline, and coordination problems. Fever and headache are usually absent. Diagnosis is based on MRI findings and detection of JC virus DNA in cerebrospinal fluid by PCR. Treatment focuses on immune

restoration, particularly with effective antiretroviral therapy.

Clinical and Public Health Implications:

HIV/AIDS affects both central and peripheral nervous systems at all stages, causing complications such as HIV-associated neurocognitive disorder, encephalitis, myelopathy, peripheral neuropathy, and opportunistic infections. Patients may develop cognitive decline, seizures, behavioral changes, and motor or sensory deficits, leading to increased morbidity, mortality, and poor treatment adherence. Neurological disability contributes to healthcare burden, productivity loss, and social dependency, particularly in low-resource settings where stigma and limited access to care persist. Early diagnosis, routine neurological assessment, and prompt antiretroviral therapy are vital. Public health efforts should promote testing, universal ART access, adherence support, rehabilitation, and integrated neurological care

Conclusion

Patients living with HIV infection are more prone for opportunistic infections and malignancy which are seen rarely in general population. CD4 count plays significant role in prevention of these opportunistic infections. Higher the CD4 count, lesser is the chance of developing the opportunistic infections and better is the prognosis.

Early diagnosis of retroviral disease and prompt treatment with good compliance reduces the chances of acquiring the disease. High risk population like drug abusers, health care workers, prostitutes etc. should be screened regularly for detection HIV infection and to prevent further occurrence of opportunistic infections.

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