



# Anti-N-methyl-D-aspartate Receptor Encephalitis in People Living with HIV: Case Report and Literature Review

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## ABSTRACT

With the increase in the number of cases of autoimmune encephalitis (AE), the cerebrospinal fluid (CSF) of people living with HIV (PLWH) showing abnormal behavior, cognitive impairment or abnormal movements should be actively screened for the antibody panel of AE. Early

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recognition and treatment can prevent severe seizures or coma and markedly improve the prognosis of patients. The first-line immunotherapy for AE includes intravenous methylprednisolone and immunoglobulin. However, whether long-time immunosuppressive maintenance therapy is needed is debated. For PLWH, immunosuppressive therapy and even steroids could be more challenging. Here, we review and summarize the clinical characteristics often reported cases and report one case from our center to improve the diagnosis and treatment of anti-N-methyl-D-aspartate receptor encephalitis in PLWH.

**Keywords:** PLWH; Anti-NMDAR encephalitis; Case report; Literature review

## Key Summary Points

For people living with HIV (PLWH) with abnormal mental behavior, cognitive or motor disorders, the antibody spectrum of autoimmune encephalitis in cerebrospinal fluid (CSF) should be actively screened for early diagnosis

Cytokine storm in CSF may be related to anti-NMDA receptor encephalitis

Dynamic monitoring of CSF cytokines helps guide immunotherapy and evaluate treatment efficacy

## INTRODUCTION

Encephalitis is a serious inflammatory disease of the brain that can have infectious or autoimmune etiology. The most common causes of encephalitis are infectious factors [1]. The number of cases of autoimmune encephalitis has been gradually increasing in recent years [2, 3]. Approximately 21% of encephalitis cases in Northern Europe were autoimmune in etiology, and the most common disease was anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis. Furthermore, the disability and mortality rates were higher in patients with an impaired immune system [4]. Anti-NMDAR encephalitis is an acute and subacute brain inflammation thought to be caused by anti-NMDAR [1]. For people living with HIV (PLWH), the interaction and complexity between HIV infection and autoimmune disease are still unclear. Infection with herpes simplex virus (HSV) and other neurogenic viruses have been reported to be the potential triggers of anti-NMDAR encephalitis [5–7]. However, whether the HIV is associated with the anti-NMDAR encephalitis and specific underlying pathogenic mechanisms remain unknown. Compared to the general population, the clinical symptoms of anti-NMDAR encephalitis in PLWH are more complex, including abnormal behavior, cognitive impairment and abnormal movements, seizures and even coma [8]. Furthermore, without early treatment, prognosis is poor. Early recognition and treatment can reduce the risk of patients progressing to seizures or coma and markedly improve patient prognosis.

Treatment of anti-NMDAR encephalitis in PLWH is more complex. It is necessary to ensure effective control of HIV replication and the progression of anti-NMDAR encephalitis. Attention also needs to be paid to the drug interaction in the treatment plan to avoid serious adverse drug reactions. First-line immunotherapy for the disease includes intravenous methylprednisolone, intravenous immunoglobulin (IVIG) and plasma exchange (PE) [9]. If the first-line treatment is ineffective, the second-line treatment mainly includes

rituximab and mycophenolate-mofetil [10]. However, whether long-time immunosuppressive maintenance therapy is needed is debated. For PLWH, immunosuppressive therapy and even steroids could be more challenging. Therefore, we review and summarize the clinical characteristics of ten reported cases and report one case from our center to improve the diagnosis and treatment of anti-NMDAR encephalitis in PLWH. We believe our findings may serve as a reference for clinical practice.

## CASE PRESENTATION

A 27-year-old man with HIV infection presented with fever, headache and progressive neuropsychiatric symptoms, including inattention, decreased consciousness, slow reaction, abnormal movements, change of sleep time, abnormal shoulder movements, and fatigue. He was treated with antiretroviral therapy (ART) (tenofovir + lamivudine + efavirenz) for 5 years with undetectable viral load and no AIDS-defining disease. The patient had a history of persistent diarrhea 3 weeks before onset. He had no other past medical history and no history of mental illness or medicine abuse.

On admission, his body temperature reached a maximum of 39 °C while his vital signs were stable. Physical examination revealed impaired attention, involuntary twitching of the mouth and face, abnormal shoulder movements and myoclonus of both lower limbs. HIV RNA was not detected in plasma, and CD<sup>+</sup> 4 T cell count was 694/ul. The blood cell count, liver and kidney function, and thyroid function test results were normal. Bacteria, fungi, mycoplasma and viruses also tested negative. A lumbar puncture was then performed, and the intracranial pressure was 350 mmH<sub>2</sub>O. Cerebrospinal fluid (CSF) analysis showed increased nucleated cells ( $87 \times 10^6/l$ ) and raised inflammatory cytokines (Table 1). Unbiased metagenomic next-generation sequencing was performed to detect occult pathogens from the CSF sample, and no evidence of pathogen infection was found. This included PCR testing for HSV-1, HSV-2, Epstein-Barr virus (EBV), varicella-zoster virus (VZV), John Cunningham

**Table 1** Changes in cerebrospinal fluid indexes during treatment

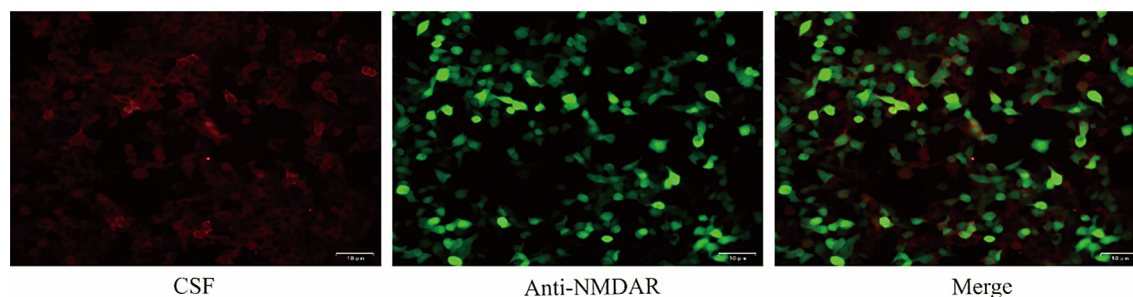
Time	Intracranial pressure mm/H <sub>2</sub> O	Nucleated cells (10 <sup>6</sup> /l)	Protein (g/l)	IL-6 (pg/ml)	IL-8 (pg/ml)	IL-10 (pg/ml)	IL-12P70 (pg/ml)	TNF- $\alpha$ (pg/ml)
Day 0	350	87	0.25	549.24	299.34	9.09	10.93	10.48
Day 5	Intravenous methylprednisolone and immunoglobulin							
Day 7	330	31	0.21	159.86	136.08	4.89	6.69	6.31
Day 12	170	19	0.26	18.5	87.68	5.23	5.61	6.70
Day 30	220	7	0.14	32.83	83.12	4.57	5.18	5.79
Day 60	160	8	0.21	14.90	111.17	5.44	5.44	6.75

virus, cytomegalovirus, enterovirus, parechovirus and human herpes viruses (HHV6), cryptococcal antigen, mycobacterium culture, nucleic acid amplification test (GeneXpert) and bacterial culture. The results of these tests were negative. Electroencephalogram (EEG) was slightly abnormal without epileptiform changes. Cerebral magnetic resonance imaging (MRI) revealed an abnormal signal in the left frontal cortex on T2-weighted fluid-attenuated inversion recovery (FLAIR) image (Figure S1). Luckily, the presence of NMDAR antibodies was identified in the CSF, whereas absence of other antibodies commonly linked to autoimmune encephalitis was observed (Fig. 1). No evidence of tumor was detected by ultrasound and computed tomography examination in the subject. In terms of differential diagnosis, brain tissue biopsy is the gold standard for the diagnosis of CD8 encephalitis, but due to its invasiveness and risks, it is usually only considered when other non-invasive tests cannot confirm the diagnosis. In addition, cognitive dysfunction caused by efavirenz appears earlier or persists, and patient symptoms may improve after discontinuing the drug or reducing the dosage. However, the patient had been using antiviral therapy for 5 years and HIV virus was well controlled. The patient's symptoms improved

after receiving hormone combined immunotherapy. Therefore, the patient was diagnosed with autoimmune encephalitis.

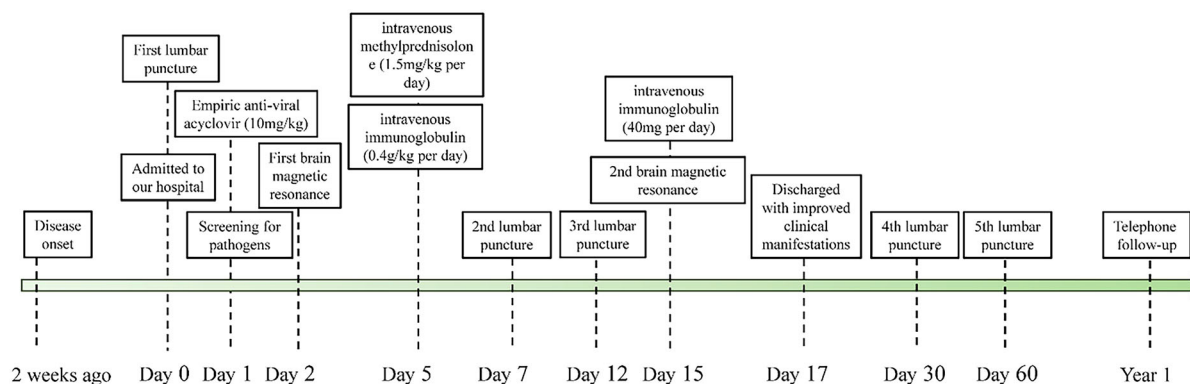
A simplified diagnosis and treatment schema for the patient is shown in Fig. 2. The patient was treated with 5 days of intravenous methylprednisolone (1.5 mg/kg/day) and intravenous immunoglobulin (0.4 g/kg/day) followed by a tapering regimen of methylprednisolone (initial amount 40 mg daily) for 6 months. After 5 days, the patient started to show clinical improvement, and CSF examination revealed a significant decrease in nucleated cells and inflammatory cytokines. There were no headaches or fever, and no abnormal movements after 28 days. With apparent remission according to neuroimaging and CSF analysis, the patient was discharged and received regular follow-ups. During the follow-up period, the CSF examination was done several times to assess cytokine levels to facilitate medication adjustments (Table 1). The patient was stable with no recurrence after 1 year of follow-up.

Informed consent was obtained from the patient for publication of this case report. This study was performed in accordance with the Helsinki Declaration of 1964 and its later amendments. This study was approved by the



**Fig. 1** Anti-*N*-methyl-D-aspartate receptor as an antigen of patient CSF autoantibodies: confirmation by a cell-based assay that HEK-293 cells were transfected with the pCMV6-XL5-AK5 plasmid for transient overexpression. Fixed and permeabilized cells were then immunostained

with the patient's CSF and a commercial anti-NMDAR antibody, respectively. Total colocalization between CSF and anti-NMDAR staining was observed. Scale bar = 10  $\mu$ m



**Fig. 2** Schematic illustration for the diagnostic and managements of our patient

Institutional Review Committee of the First Affiliated Hospital of Zhejiang University School of Medicine (no. IIT20220194B-RI).

## DISCUSSION

Currently, the publicly reported incidence rate of HIV-positive anti-NMDAR encephalitis is low and misdiagnosis or delayed diagnosis of the condition is common in clinical practice. We reviewed ten previously reported cases [2, 3, 5, 11–14] and one case from our center and summarized the clinical characteristics of the disease (Table 2).

Anti-NMDAR encephalitis mostly occurs in children and young women. The search for an

underlying tumor, particularly ovarian teratoma in women or testicular germ-cell tumor in men, is of paramount importance in the context of an anti-NMDAR encephalitis [3]. Most patients have prodromal symptoms, such as headache, fever and flu-like symptoms. Children often show behavioral abnormalities and abnormal movement, while adults have mental symptoms and seizures. Approximately 35% of patients have brain MRI abnormalities [8]. A literature review showed that among PLWH, anti-NMDAR encephalitis developed mainly in young and middle-aged men (7/10), and no tumors were found (10/10). PLWH may exhibit more complex and severe clinical manifestations due to the coexistence of HIV infection and other opportunistic infections. The

**Table 2** Published cases of anti-NMDAR encephalitis in patients with HIV infection

Sex/ age/country	ART time/ ART regimen adjustment during hospitalization	CD4+ T cell count, cells/ mm <sup>3</sup> ; sample: HIV RNA, copies/ml	Clinical symptom	CSF	Sample: subunit target (NMDAR titers)	Brain MRI EEG	Immunotherapy	Outcome
Cunill V, 2016 (14)	26 Years/no adjustment	> 400/Serum: < 200 CSF: UN	②④⑤	OCB	Serum: - CSF: +	Normal/UN	MTP	UN
Cunill V, 2016 (14)	12 Years/ adjustment	899/UN CSF: UN	①	UN	Serum: + CSF: UN	Brain atrophy/ normal	UN	VL: undetectable
Patarata E 2016(3) Portugal	16 Years/UN	Normal/serum: undetectable CSF: undetectable	①③④⑤⑥⑦	WBC 86/mm <sup>3</sup>	Serum: + (1:80) CSF: + (1:8)	T2 abnormal/ diffuse slow electrogenesis	MTP (1 g/day) + IVIG(0.2 g/kg) 5-day + PE (12 sessions); RTX:(375 mg/mm <sup>3</sup> /week, 4-week) + GPH(15 mg/ kg/month, 3-month) + MMF(250 mg- 500 mg-1000 mg bid per day)	24 Months/normal motor state; moderate cognitive and memory impairment
Arboleya S 2016 (10)	3 Years/UN	Normal/ serum: < 200 CSF: UN	①⑦	UN	Serum: + (1:20) CSF: UN	Brain atrophy/ normal	UN	18 Months/ neurological examination and EEG were normal; MRI: brain atrophy
Fan CF, 2017 (12)	UN/ switch to increase CNS penetration	Un/serum: < 200 CSF: 15,800	①③④⑥	UN	+	T2-FLAIR abnormal/ normal	Steroid hormone	24 Months/near- complete recovery
Haneche F 2018 (2) Caucasian	8 Years/ adjustment	433/Serum: 97,156 CSF: 37,755	①②④⑤⑦	WBC 48/mm <sup>3</sup>	Serum: UN CSF: +	T2-FLAIR abnormal/theta- delta waves	IVIG	Cognitive and behavioral recovery. EEG showed improvement. Brain MRI was unchanged. (CSF) NMDAR -

Table 2 continued

Sex/ age/country	ART time/ ART regimen adjustment during hospitalization	CD4+ T cell count, cells/ mm <sup>3</sup> ; sample: HIV RNA, copies/ml	Clinical symptom	CSF	Sample: subunit target (NMDAR titers)	Brain MRI EEG	Immunotherapy	Outcome
Anguizola- Tamayo D 2019(11)	29 Years/ adjustment	628/Serum: 60 CSF: 45,600	①④⑥	WBC 35/mm <sup>3</sup>	Serum: UN CSF: + (1:160)	T2-FLAIR abnormal/ diffuse slowing	MTP (500 mg, 5-day) IVIg (0.4 mg/kg, 5-day)	7 Months/MRI showed improvement and diffuse white matter persisted
Pinzon- Charry, 2019 (13)	2 Years/no	> 500/Serum: undetectable CSF: undetectable	①④⑤⑦	OCB	Serum: + CSF: +	Brain atrophy/UN	IVIg (1 g/kg, 12-month) + MTP (30 mg/kg, 3-month); IVIg (1 g/ kg, 12-month) + MTP (30 mg/ kg, 4-month)	24 Months/ recurrence: 1 time, mild cognitive and memory impairment; normal CD4 T-cell count; HIV suppression
Moloney PB, 2020 (5)	3 Years/switch to increase CNS penetration	180/Serum: 916 CSF: 42,365	①④⑤	WBC 160/mm <sup>3</sup>	Serum: + CSF: +	T2-FLAIR abnormal/ triphasic complexes	Steroid hormone 3-day IVIg 10-day	2 Months/(CSF) NMDAR-, MRI EEG: normal VL: undetectable

**Table 2** continued

Sex/ age/country	ART time/ ART regimen adjustment during hospitalization	CD4+ T cell count, cells/ mm <sup>3</sup> ; sample: HIV RNA, copies/ml	Clinical symptom	CSF	Sample: subunit target (NMDAR titers)	Brain MRI EEG	Immunotherapy	Outcome
Moloney PB, 2020 (5)	M/41/ African American	878/Serum: 31 CSF: 15,800	①②③④⑤⑥⑦	WBC 105/mm <sup>3</sup>	Serum: + CSF: +	T2-FLAIR abnormal/ normal	MTP + PDN; MTP + RTX	48 Months/ recurrence: 2 times, inability to care for themselves, cognitive and mental improvement

F female, M male, ART antiretroviral therapy, VL viral load, CNS central nervous system, NMDAR N-methyl-D-aspartate receptor, WBC white blood cell count, OCB oligoclonal bands, EEG electroencephalogram, MRI magnetic resonance imaging, D day, M month, CPH cyclophosphamide, IVIG intravenous immunoglobulin, MTP intravenous methylprednisolone, PDN oral prednisone, PEX plasma exchange, RTX rituximab, UN unknown

a: According to the diagnostic criteria (8), the clinical manifestations of anti-NMDAR encephalitis were as follows:

- ① Abnormal (psychiatric) behavior or cognitive dysfunction; inattention, irritability, anxiety, nonsense, hallucination, delusion, thinking disorder, self-talk
- ② Speech dysfunction (pressured speech, verbal reduction, mutism): verbal vagueness, imitation of speech, speech apraxia, repetition of speech
- ③ Seizures
- ④ Movement disorder, dyskinesias or rigidity/abnormal postures: oral and facial dyskinesia, abnormal limb movements, Parkinson-like symptoms, muscle weakness, myoclonus, gait abnormalities
- ⑤ Decreased level of consciousness: decline of consciousness, slow reaction, disorientation, anterograde amnesia, memory impairment
- ⑥ Autonomic dysfunction or central hypoventilation, mechanical ventilation, coma
- ⑦ Others: sleep rhythm change, systemic myalgia, anorexia

most common clinical manifestations of the disease were abnormal behavior and movement disorders. MRI abnormalities were more common (9/10), and EEG abnormalities were rare. A young male patient in our center presented with behavioral abnormalities and autonomic dysfunction. Ultrasound and CT of the body showed no tumors in our patient. An abnormal signal was detected on T2-weighted FLAIR MRI, and EEG showed a slight abnormality without epileptiform changes.

An early study showed that the sensitivity and specificity of antibody detection in the CSF of patients with anti-NMDAR encephalitis were very high, while false-positive or -negative results could be observed in the serum. Approximately 14% of patients with anti-NMDAR encephalitis had antibodies in the CSF but none in the serum [8, 15]. Therefore, it is important to actively screen for the antibody panel of autoimmune encephalitis in cerebrospinal fluid for the diagnosis of anti-NMDAR encephalitis.

Previously, HSV and other neurotropic viruses were thought to be potential triggers of anti-NMDAR encephalitis [7, 8]. It was hypothesized that HSV-induced inflammation and destruction may expose neuronal antigens, thereby triggering humoral autoimmunity. However, the relationships between HIV and autoimmune encephalitis remain poorly defined. Prior reports suggest that HIV may contribute to the generation of NMDAR antibodies. Review of ten prior cases showed that 50% of these patients have high CSF HIV viral loads. It was found that NMDAR was activated by HIV-1 envelope glycoprotein 120 (gp120), which led to neuronal damage after damage to the blood-brain barrier [16]. In addition, increased phosphorylation of NMDAR receptor subunits was detected in human brain tissues with HIV encephalitis infection [17], which could be attributed to HIV Tat protein. All these findings indicate that HIV infection may trigger anti-NMDAR encephalitis; however, the specific mechanism remains to be elucidated. Additionally, the case in our center suggests anti-NMDAR encephalitis occurs despite well-controlled HIV in CSF and plasma, in line with previous studies [2, 13] implying that other factors besides HIV infection are

involved in the development of anti-NMDAR encephalitis. Furthermore, because of the patient's history of persistent diarrhea 3 weeks before onset in our center, herpes virus infection could not be ruled out, which then led to an immune response disorder to the opportunistic pathogen. It was observed in our case that white blood cell counts and levels of TNF- $\alpha$ , IL-6, IL-8, and other cytokines in the CSF significantly increased, indicating a potential role of inflammatory cytokine storms in the development of anti-NMDAR encephalitis, which is supported by studies done elsewhere [18].

Once diagnosed, the extraordinary complexity of anti-NMDAR encephalitis therapy remains a major challenge. It is necessary to ensure effective control of HIV replication and the progression of anti-NMDAR encephalitis. Literature review found that 6/10 patients adjusted the ART regimen during hospitalization. Among them, four subjects had low viral load in serum but high viral load in CSF. To reduce the viral load in CSF, they switched to an ART regimen with increased central nervous system (CNS) penetration; the ART regimen was adjusted in another patient because of poor control of HIV in serum and cerebrospinal fluid. The last patient switched his ART regimen but detailed information was not given.

First-line immunotherapy for the condition includes intravenous methylprednisolone and IVIG or plasma exchange [9]. To reduce the risk of intracranial inflammatory reaction and recurrence, a corticosteroid was recommended for maintenance therapy.

Because of their impaired immune status, the patients' treatment response and prognosis for anti-NMDAR encephalitis in PLWH may differ from those of the general population, requiring extra caution during treatment. For PLWH, a long course of high-dose corticosteroid therapy could further inhibit impaired immunity, increasing the risk of opportunistic infections and complexity of the condition [19].

In our case, during maintenance therapy, the amounts of corticosteroid were adjusted in time according to the levels of cytokines IL-6, IL-8, and TNF- $\alpha$ . After 2 months, there were no behavioral abnormalities. Additionally, intracranial pressure and levels of inflammatory



cytokine decreased, the nucleated cell count normalized and anti-NMDAR antibody levels in CSF became negative again. Therefore, considering the predictive role of cytokines in the CSF, it was recommended that cytokine levels in the CSF should be checked periodically to guide immunotherapy and assess efficacy.

## CONCLUSION

Anti-NMDAR encephalitis in PLWH can be easily confused with mental diseases, leading to incorrect or delayed diagnoses. It is associated with a high disability rate and seriously affects the quality of life. Therefore, it is necessary to actively screen the antibody panel of autoimmune encephalitis in the CSF for an early diagnosis. In addition, dynamic monitoring of CSF cytokines helps guide immunotherapy and evaluate treatment efficacy.

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**Author Contributions.** Jiangjin Hui wrote the manuscript; Jinhua Wang and Zhikai Wan collected the basic information of the patients and edited the manuscript; Qing Cao, Bohao Dai and Haiyan Lou were responsible for imaging data and processing; Biao Zhu reviewed and edited the manuscript. All authors read and approved the final manuscript.

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**Data Availability.** All data generated or analyzed during this study are included in this published article and supplementary information files.

### Declarations

**Conflict of Interest.** Jiangjin Hui, Qing Cao, Zhikai Wan, Bohao Dai, Haiyan Lou and Biao Zhu have no relevant financial or non-financial interests to disclose.

**Ethical Approval.** Informed consent was obtained from the patient for publication of this case report. This study was performed in accordance with the Helsinki Declaration of 1964 and its later amendments. This study was approved by the Institutional Review Committee of the First Affiliated Hospital of Zhejiang University School of Medicine (no. IIT20220194B-RI).

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