

CASE REPORT

Open Access



Time moving 100-fold slower: time distortion as a diagnostic clue in anti-NMDA receptor encephalitis

Risa Hirata^{1*}, Hisashi Wada¹, Kazunori Yamamoto¹, Yuji Sogi¹, Hiroto Muzuta¹, Yu Isaka¹ and Michitaka Funayama²

Abstract

Background The primary symptoms in the early stages of anti-NMDA receptor encephalitis are psychiatric manifestations, making it difficult to distinguish from psychiatric disorders. While anti-NMDA receptor encephalitis requires a completely different treatment approach, the specific psychiatric features of the condition remain poorly identified. Although previous studies have suggested that altered perceptions may be characteristic, few case reports focus on altered perceptions of time or time distortion, a phenomenon closely linked to NMDA receptor dysfunction as seen in individuals using NMDA receptor inhibitors like ketamine and phencyclidine. In this report, we describe two cases of anti-NMDA receptor encephalitis manifesting pronounced time distortion in its early stages, which may serve as diagnostic clues for the early diagnosis and treatment of this potentially lethal condition.

Case presentations Two cases of Anti-NMDA receptor encephalitis, both marked by significant time distortion in the early stages and showing near-complete recovery with immunotherapy, are presented in detail. In both cases, time distortion was the predominant symptom among the psychiatric manifestations. Case 1: A middle-aged man experienced a pronounced perception of time moving 100 times slower in the early stages, accompanied by feelings of detachment and auditory abnormalities. This time distortion persisted for over a year, even after other symptoms had fully resolved. Case 2: A young woman reported that time seemed to move two to three times slower in the early stages. Although she did not initially mention time distortion, she confirmed it when specifically questioned.

Conclusions Our report suggests that time distortion, particularly the perception of time moving slowly, can be a distinguishing feature in the early stages of anti-NMDA receptor encephalitis. This unique characteristic, especially when occurring independently of other symptoms, is rare as a primary and isolated symptom in other conditions, making it useful for differentiation from time distortion in other psychiatric disorders. Additionally, since some patients may not spontaneously report time distortion, actively assessing this symptom during early evaluation could help improve diagnostic accuracy.

Keywords Anti-NMDA receptor encephalitis, Time distortion, Altered sense of time, Altered perception, Slow-motion perception

*Correspondence:

Risa Hirata
risahirata525@gmail.com

¹Department of Neuropsychiatry, Osaka Red Cross Hospital, 5-30
Fudegasakicho, Tennoji-ku, Osaka 5438555, Japan

²Department of Neuropsychiatry, Ashikaga Red Cross Hospital, 284-1,
Yobe, Ashikaga-City 3260843, Tochigi, Japan



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

Background

Anti-NMDA receptor encephalitis is a serious, life-threatening disease, with 29% of patients reportedly admitted to the ICU [1] and 5–11% dying from the condition [2–4]. The prognosis of autoimmune encephalitis largely depends on rapid diagnosis and the early initiation of therapy. Early immunotherapy results in substantial recovery in 70–80% of the patients [1, 5–7]. However, rapidly differentiating anti-NMDA receptor encephalitis from psychiatric disorders, particularly schizophrenia spectrum disorders, can be challenging. This is because anti-NMDA receptor encephalitis often presents with psychiatric symptoms in the early stages, without obvious neurological findings [5, 8–12]. Reports indicate that 87% of patients with anti-NMDA receptor encephalitis experience acute behavioral changes, and 59% initially present with psychiatric symptoms. Among those with psychiatric symptoms, as many as 87% meet criteria for cycloid psychosis [13], which resembles both schizoaffective disorder and brief psychotic disorder. Specifically, 23% meet criteria for acute schizoaffective disorder [12], and 40% experience visual or auditory hallucinations [12]. The similarity of these psychiatric symptoms to common psychiatric disorders, such as schizophrenia spectrum disorders, often leads to misdiagnosis in the early stages of anti-NMDA receptor encephalitis [14–16]. As a result, up to 77% of patients first visit a psychiatrist [9], and 31% are initially admitted to psychiatric wards [2, 3]. In addition, laboratory findings, such as magnetic resonance imaging (MRI), often show no abnormalities in up to 70% of affected individuals [13], and even after spinal fluid testing is performed, the definitive diagnosis cannot be made immediately because it takes some time before the spinal fluid is positive for anti-NMDA receptor antibodies [17]. Despite the significant challenges in diagnosis, a good prognosis in anti-NMDA receptor encephalitis depends on early treatment [3, 18–21]. What is needed are specific psychiatric symptoms that differentiate anti-NMDA receptor encephalitis from other psychiatric disorders, which could significantly improve the prognosis, particularly when neurological symptoms are absent and laboratory findings are either unavailable or inconclusive in the early stages.

While previous studies have suggested that altered perception is a characteristic symptom of anti-NMDA receptor encephalitis [5, 22], the specific types of these alterations remain unclear. Time distortions, in particular, may be linked to this encephalitis, as drugs that inhibit NMDA receptor function, like ketamine and phencyclidine [23–26], commonly cause prominent time distortion among other perceptual disturbances. In fact, time distortion is one of the most consistently reported effects of these drugs [23–26]. A study found that 11 out of 15 healthy volunteers using ketamine complained

of time distortion, mostly in slow-motion perceptions [26]. Since antibodies inhibit NMDA receptor function in anti-NMDA receptor encephalitis, this may lead to similar symptoms [27, 28]. Despite these potentially significant connections, only one case report has mentioned time distortion, describing it as an additional feature among various types of perceptual alterations in this condition [29].

We report two patients with anti-NMDA receptor encephalitis who exhibited pronounced time distortion, a novel finding as it emerged as a characteristic symptom during the acute phase. We tracked the progression of this symptom throughout the course of the encephalitis and compared it to time distortion in other psychiatric disorders as well as in other types of encephalitis. Our observations offer new insights into the psychiatric features of anti-NMDA receptor encephalitis and may aid in its early diagnosis, even when detailed examinations such as MRI or cerebrospinal fluid analysis are unavailable, particularly in psychiatric outpatient settings. This can be achieved at no additional cost and may lead to timely treatment of this life-threatening but often difficult-to-diagnose condition.

Case presentations

Ethical aspects of this study were reviewed and approved by the Osaka Red Cross Hospital Human Research Ethics Committee. Between June 2023 and June 2024, two patients with anti-NMDAR encephalitis were admitted to the psychiatry unit of Osaka Red Cross Hospital. Both patients experienced time distortion, prompting this report. The patients and their family members granted informed consent in accordance with the Declaration of Helsinki. The definitive diagnosis of the two patients was based on diagnostic criteria for anti-NMDA receptor encephalitis [17] and the presence of anti-human IgG anti-GluN1 antibodies in cerebrospinal fluid as assessed with enzyme-linked immuno-sorbent assays. Both patients were primarily treated by psychiatrists and neurologists and were followed until full recovery in our psychiatry unit. This setting allowed for a thorough assessment of psychiatric symptoms, which might not have been as feasible in a neurology unit. In both cases, a psychiatrist with more than 35 years of clinical psychiatric experience and several younger psychiatrists worked together in the treatment of the patients. Nursing care was provided by psychiatric nurses, and rehabilitation was performed by an occupational therapist specialized in psychiatry. During the inpatient treatment period, the psychiatrist inquired in detail at least once a week about the patients' time distortion, including any associated abnormalities in modalities such as visual or auditory perception. In Case 1, post-discharge outpatient visits

were held once a month, with the psychiatrist checking for the time distortion on each visit.

Case 1

Case 1 is a 40-year-old man with a history of depression triggered by interpersonal relationship problems at work two times, at 26 and 35 years of age. Forty-three days before admission, he developed a headache and a fever of around 38 °C, which subsided on its own, though a mild fever persisted. Three days before admission, he developed altered perceptions and related symptoms, including a distorted sense of time, a perception of the world in sepia tones, and a feeling of disconnection from reality. As these symptoms intensified, he developed suicidal thoughts and started sleeping only about one hour per night. His sense of time moving slowly gradually worsened. One day before admission, this sensation intensified tenfold, along with a rise in anxiety. On the day of admission, his perception of time felt as it had slowed by up to 100-fold. He made an emergency visit to the neurology department of our hospital. He was admitted to our inpatient psychiatry unit, which provides a more secure environment for individuals with suicidal ideation. Neurological signs observed upon admission included involuntary movements in both upper limbs and altered consciousness, with a Glasgow Coma Scale score of E4, V4, and M6 [30].

He was diagnosed with autoimmune encephalitis, including anti-NMDAR encephalitis, based on preceding fever, psychiatric symptoms atypical for psychiatric conditions such as schizophrenia, altered consciousness, cerebrospinal fluid analysis showing 154 cells/ μ L with 97% monocytes, and a faint high signal in the left temporal-occipital-parietal cortex on the Fluid Attenuated Inversion Recovery (FLAIR) MRI. His electroencephalogram (EEG) revealed no abnormalities and contrast-enhanced computed tomography (CT) of his trunk showed no findings suggestive of a seminoma or other tumors in the testis. The diagnosis of anti-NMDA receptor encephalitis was confirmed when elevated anti-human IgG anti-GluN1 antibodies were subsequently detected in his cerebrospinal fluid.

In his clinical course, the peak occurred approximately two weeks after admission, marked by worsening neurological and autonomic nervous signs. By hospital day 12, his muscle stiffness and involuntary movements in both upper limbs intensified, accompanied by increased sweating and tachycardia, eventually leading to unresponsiveness with a GMS score of E2, V2, and M4. Central hypoventilation was not observed. He was treated with intensive immunotherapy, including four cycles of steroid pulse therapy (1000 mg of methylprednisolone per day for 3–5, 22–24, 29–31, and 36–38 hospital days after admission), intravenous immunoglobulin once

(400 mg/kg of human immunoglobulin, 8–12 hospital days after admission), and intravenous cyclophosphamide twice (1500 mg on hospital days 16 and 43). After hospital day 18, his consciousness gradually improved, with a GMS score of E4, V4, and M6. Around one month post-admission, his involuntary movements, sweating, and tachycardia resolved, and his consciousness further improved.

For his psychiatric symptoms, he exhibited a strong desire to die and significant agitation upon admission, prompting the initiation of antipsychotic risperidone at 3 mg, which was gradually increased to 8 mg by the end of the day. On hospital day 4, his behavior became increasingly chaotic; he violently banged on his room door and repeatedly said, “there is someone else in me.” He was additionally prescribed the mood stabilizer valproic acid at 400 mg and the antipsychotic zotepine at 100 mg. Despite these interventions, around hospital day 10, he remained markedly disoriented and chaotic, incorrectly identifying his location as “bank” and his age as “1020 years old.” He also experienced time distortion, describing time as moving extremely slowly, and reported auditory perception abnormalities, such as hearing nurses speak slowly. However, this time distortion was not accompanied by visual or kinesthetic perception abnormalities.

When his consciousness became alert again during the recovery phase after passing the peak of his clinical course, he became agitated again, exhibiting aggressive behaviors such as hitting nurses and attempting to strangle himself with the intravenous line. Around one month post-admission, his agitation and aggressiveness resolved, allowing him to engage in simple conversations. However, he continued to experience cognitive dysfunctions, such as difficulty tying shoelaces, performing simple addition, and recalling the content of the previous day’s conversation. From around hospital day 35, he regained orientation and no longer reported thoughts of death. He also regained the ability to perform calculations. The sensation of time moving slowly improved from a severe 1000-fold distortion to a 2-fold distortion. By hospital day 60, the abnormal sense of time had diminished, and he only experienced significant time distortion during periods of excess free time. He was discharged with normalized cognitive function, as indicated by a Mini-Mental State Examination score of 29 [31], on hospital day 74. The amount of psychotropics was reduced after the peak of the disease and discontinued shortly after discharge. He returned to his clerical job 9 months after discharge, with a Modified Rankin Scale score of 1, indicating no significant disability [32, 33]. However, 10 months post-discharge, he still reported that time seemed to slow down to twice its usual rate when he felt stressed.

Case 2

Case 2 features a 28-year-old office worker with no prior history of psychotic disorders or family mental health issues. She had not encountered any notable stressors before the onset of her symptoms. Twenty-six days before her admission to our hospital, she began experiencing auditory disturbances, describing strange sounds in her ears, distant voices, and difficulty comprehending speech during a social outing in a flower viewing festival. The following day, she independently visited a neurology clinic, but no abnormalities were detected. Nineteen days before admission, she experienced panic over a strange taste in her food, and her condition progressively worsened. She became unable to hold coherent conversations, wandered aimlessly around the house, crawled on all fours in circles, and her thoughts, speech, and actions became increasingly chaotic. Nine days before her admission to our hospital, she was admitted to a psychiatric hospital with suspected psychiatric condition, when she became unresponsive to external stimuli. After her admission to the psychiatric hospital, she developed a fever of approximately 38 °C, which gradually subsided. However, there was no improvement in her psychiatric symptoms despite treatment with an 80 mg antipsychotic blonanserin patch. Nine days after her initial psychiatric admission, she was referred to our department to investigate the possibility of an underlying neurologic condition.

Upon arrival, she was catatonic and uncommunicative, with frequent involuntary movements in both upper limbs. Her EEG showed an extreme delta brush, although screening laboratory tests, brain MRI, and abdominal MRI (for detecting an ovarian teratoma) revealed no abnormalities. The cerebrospinal fluid cell count was slightly elevated at 10 cells/ μ L, with 90% of these cells being monocytes. Given her sudden onset of psychiatric symptoms without a prior psychiatric history or identifiable stressors, significant involuntary movements, slow waves on the EEG, and cerebrospinal fluid findings, she was diagnosed with autoimmune encephalitis, including anti-NMDA receptor encephalitis. The diagnosis of anti-NMDA receptor encephalitis was confirmed when elevated anti-human IgG anti-GluN1 antibodies were detected in her cerebrospinal fluid. She received steroid pulse therapy twice: 1000 mg of methylprednisolone per day, administered on 2–6 and 13–17 hospital days. By hospital day 5, she began responding to calls with nodding and was able to eat steadily. The involuntary movements in her upper limbs gradually subsided.

Around hospital day 10, her disorientation improved. She reported, ‘Before I was hospitalized in the psychiatric facility, my sense of time felt about 2 or 3 times slower, but now it’s back to normal.’ This suggested that during the initial phase, when she experienced auditory distortions and taste abnormalities, she was also experiencing

time distortion, specifically perceiving time in slow motion. Her altered sense of time was not accompanied by visual or kinesthetic sensory abnormalities. Additionally, she had memory impairment, such as difficulty recalling the lyrics to a song she was previously familiar with, but this improved over time. By discharge on hospital day 29, the only remaining symptom was mild insomnia, and she had a Modified Rankin Scale score of 1, indicating no significant disability.

Discussion and conclusions

We report, for the first time, two cases in which time distortion was a pronounced symptom in the early stages of anti-NMDA receptor encephalitis. This finding aligns with effects observed in drugs that inhibit NMDA receptor function, such as ketamine and phencyclidine [23–26], and may help differentiate this condition from other psychiatric disorders. In Case 1, time distortion remained the primary symptom throughout the initial and residual phases, lasting nearly one year. In Case 2, although time distortion appeared in the early stages, the patient was confused and presumably unable to convey her experience clearly. These findings suggest that clinicians should actively assess for time distortion, especially when patients with anti-NMDA receptor encephalitis are confused and unable to articulate their specific symptoms due to prominent psychiatric disturbances.

We review the characteristics of time distortion based on our two cases and two additional cases from a previous report on altered perception in anti-NMDA receptor encephalitis [29]. The most striking feature is that, in three of the four cases with time distortion (75%), patients experienced the sensation of time moving slowly. Additionally, out of the four cases, two featured altered perceptions in visual and auditory modalities, respectively.

Although time distortion occurs in other conditions, including schizophrenia and other forms of encephalitis [34, 35], slow-motion perception is less common. A previous study of 84 patients with time distortion, regardless of their underlying condition, found that 29 (34.5%) experienced slow-motion perception [34, 35]. Another study reported that, out of 301 patients with schizophrenia, 109 experienced time distortion, but only 12 (4.0%) reported perceiving the sensation of time moving slowly [36]. In schizophrenia, time distortion is often characterized by a fragmented sense of time, such as viewing the world “like a series of photographs,” and is associated with disorganization, which is a hallmark of the disorder [36–38]. This abnormality is also described as bizarre alterations in time perception rather than slow-motion perception [39, 40]. In contrast, none of the four cases of anti-NMDA receptor encephalitis showed the time fragmentation typical of schizophrenia. Furthermore,

Case 1 of anti-NMDA receptor encephalitis reported extreme symptoms, including perceiving time as moving 100 times slower, a phenomenon rarely observed in schizophrenia.

We also compare the time distortion of anti-NMDA receptor encephalitis with that of other types of encephalitis. Most of the reported cases of time distortion in other types of encephalitis are associated with Alice in Wonderland Syndrome (AIWS), which is a perceptual disorder characterized by distortions of visual perception, the body schema, and time [41]. However, in most types of encephalitis with AIWS, distortions of visual perception and the body schema are common and time distortion is rare [42]. For example, among Epstein-Barr virus (EBV) encephalitis, which is the most common disease that causes AIWS, only two cases have been reported with time distortion, and even in both of those cases, metamorphopsia, a defect of vision in which objects appear to be distorted, was more prominent than time distortion [43, 44]. In other encephalitis, one case of slow-motion perception has been reported in influenza encephalitis, which was also prominent with metamorphopsia [45]. In summary, other encephalitis is often accompanied by altered perceptions, particularly metamorphopsia, and time distortion is rarely complained of almost independently, as in cases with anti-NMDA receptor encephalitis.

Altered perceptions have been found to be more common in patients with autoimmune encephalitis, including anti-NMDA receptor encephalitis, than those in schizophrenia spectrum disorders [22]. Our findings go further, suggesting that among these altered perceptions, time distortion—particularly the sensation of time moving slowly—may be a key differentiating factor between anti-NMDA encephalitis and psychiatric conditions such as schizophrenia, as well as possibly other forms of encephalitis. Confirming the presence of this symptom is straightforward and can be done even when detailed examinations such as MRI or cerebrospinal fluid analysis are unavailable, particularly in psychiatric outpatient settings. Even in cases where MRI findings are inconclusive, this symptom can serve as a suggestive sign, prompting further investigation, such as cerebrospinal fluid analysis. This approach incurs no additional cost and may facilitate the timely treatment of this life-threatening but often challenging-to-diagnose condition.

Limitation

Time distortion as a diagnostic marker in anti-NMDA receptor encephalitis remains speculative due to several limitations. First, while our discussion compares schizophrenia and anti-NMDA receptor encephalitis, recent studies suggest a continuum between anti-NMDA receptor encephalitis and psychiatric disorders associated with

NMDA receptor antibodies [46, 47]. The prevalence of time distortion symptoms, particularly in NMDA receptor antibody-positive schizophrenia, has not been thoroughly investigated. Moreover, it remains unclear at which stage of this continuum time distortion symptoms emerge. Second, the number of cases was too small to generalize the results, and our cases are subject to bias, as they involve patients admitted to a psychiatric hospital. Therefore, it is necessary to examine time distortion, particularly the experience of slow-motion perception, across all cases of anti-NMDA receptor encephalitis to accurately determine its prevalence.

Conclusion

Our report suggests that time distortion, especially slow-motion perception, is a characteristic symptom of anti-NMDA receptor encephalitis, occurring even in the early stages. Unlike schizophrenia or other forms of encephalitis, slow-motion perception is more commonly reported as a primary complaint in patients with anti-NMDA receptor encephalitis. Therefore, assessing this symptom is important, as it may help in early diagnosis and improve prognosis.

Acknowledgements

Not applicable.

Author contributions

RH drafted the initial version of the case report manuscript. MF contributed to the conceptualization of the report and provided critical revisions to the manuscript. HW served as the lead clinician for these cases and reviewed the report. KY monitored the patients from admission through to their outpatient follow-ups. YS, HM, and YI contributed to the clinical evaluation of the patients during admission and to the writing of the manuscript. All authors contributed to the article and approved the submitted version.

Funding

There were no sources of funding.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient participant (the study case) for the publication of any potentially identifiable data included in this article.

Competing interests

The authors declare no competing interests.

Clinical trial number

Not applicable.

Received: 26 September 2024 / Accepted: 10 February 2025

Published online: 24 February 2025

References

1. Nissen MS, Ørvik MS, Nilsson AC, Ryding M, Lydolph M, Blaabjerg M. NMDA-receptor encephalitis in Denmark from 2009 to 2019: a national cohort study. *J Neurol*. 2022;269(3):1618–30.
2. Ayubi E, Safiri S. Risk factors for mortality in patients with anti-NMDA receptor encephalitis; comment on data sparsity. *Acta Neurol Scand*. 2017;136(6):737.
3. Titulaer MJ, McCracken L, Gabilondo I, Armangué T, Glaser C, Iizuka T, et al. Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. *Lancet Neurol*. 2013;12(2):157–65.
4. Giri YR, Parrill A, Damodar S, Fogel J, Ayed N, Syed M, et al. Anti-N-methyl-D-aspartate receptor encephalitis in adults: a systematic review and analysis. *Neuropsychiatr*. 2024;38(2):92–101.
5. Chapman MR, Vause HE. Anti-NMDA receptor encephalitis: diagnosis, psychiatric presentation, and treatment. *Am J Psychiatry*. 2011;168(3):245–51.
6. Dalmau J, Gleichman AJ, Hughes EG, Rossi JE, Peng X, Lai M, et al. Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies. *Lancet Neurol*. 2008;7(12):1091–8.
7. Herken J, Prüss H. Red flags: clinical signs for identifying Autoimmune Encephalitis in Psychiatric patients. *Front Psychiatry*. 2017;8:25.
8. Dalmau J, Armangué T, Planagumà J, Radosevic M, Mannara F, Leypoldt F, et al. An update on anti-NMDA receptor encephalitis for neurologists and psychiatrists: mechanisms and models. *Lancet Neurol*. 2019;18(11):1045–57.
9. Al-Diwani A, Handel A, Townsend L, Pollak T, Leite MI, Harrison PJ, et al. The psychopathology of NMDAR-antibody encephalitis in adults: a systematic review and phenotypic analysis of individual patient data. *Lancet Psychiatry*. 2019;6(3):235–46.
10. Warren N, Siskind D, O’Gorman C. Refining the psychiatric syndrome of anti-N-methyl-D-aspartate receptor encephalitis. *Acta Psychiatr Scand*. 2018;138(5):401–8.
11. Gibson LL, Pollak TA, Blackman G, Thornton M, Moran N, David AS. The Psychiatric phenotype of Anti-NMDA receptor encephalitis. *J Neuropsychiatry Clin Neurosci*. 2019;31(1):70–9.
12. Sarkis RA, Coffey MJ, Cooper JJ, Hassan I, Lennox B. Anti-N-Methyl-D-Aspartate receptor encephalitis: a review of Psychiatric Phenotypes and Management considerations: a report of the American Neuropsychiatric Association Committee on Research. *J Neuropsychiatry Clin Neurosci*. 2019;31(2):137–42.
13. Giné Servén E, Boix Quintana E, Martínez Ramírez M, Guanyabens Buscà N, Muriana Batiste D, Guasp M, et al. Cycloid psychosis as a psychiatric expression of anti-NMDAR encephalitis. A systematic review of case reports accomplished with the authors’ cooperation. *Brain Behav*. 2021;11(2):e01980.
14. Komagamine T, Kanbayashi T, Suzuki K, Hirata K, Nishino S. Atypical psychoses and anti-NMDA receptor encephalitis: a review of literature in the mid-twentieth century. *Psychiatry Clin Neurosci*. 2022;76(2):62–3.
15. Subeh GK, Lajber M, Patel T, Mostafa JA. Anti-N-Methyl-D-Aspartate receptor encephalitis: a detailed review of the different Psychiatric presentations and red flags to look for in suspected cases. *Cureus*. 2021;13(5):e15188.
16. Giri YR, Korie I, Hashmi S, Parrill A, Ayed N. Anti-NMDA receptor Encephalitis masquerades as psychosis: a Case Report. *J Psychiatr Pract*. 2022;28(1):72–7.
17. Graus F, Titulaer MJ, Balu R, Benseler S, Bien CG, Cellucci T, et al. A clinical approach to diagnosis of autoimmune encephalitis. *Lancet Neurol*. 2016;15(4):391–404.
18. Finke C, Kopp UA, Prüss H, Dalmau J, Wandering KP, Ploner CJ. Cognitive deficits following anti-NMDA receptor encephalitis. *J Neurol Neurosurg Psychiatry*. 2012;83(2):195–8.
19. Byrne S, Walsh C, Hacohen Y, Muscal E, Jankovic J, Stocco A, et al. Earlier treatment of NMDAR antibody encephalitis in children results in a better outcome. *Neurol Neuroimmunol Neuroinflamm*. 2015;2(4):e130.
20. Finke C, Kopp UA, Pajkert A, Behrens JR, Leypoldt F, Wuelfel JT, et al. Structural hippocampal damage following Anti-N-Methyl-D-Aspartate receptor encephalitis. *Biol Psychiatry*. 2016;79(9):727–34.
21. Lee WJ, Lee ST, Moon J, Sunwoo JS, Byun JI, Lim JA, et al. Tocilizumab in Auto-immune Encephalitis Refractory to Rituximab: an institutional cohort study. *Neurotherapeutics*. 2016;13(4):824–32.
22. Funayama M, Koreki A, Takata T, Kurose S, Hisamatsu T, Ono A, et al. Differentiating autoimmune encephalitis from schizophrenia spectrum disorders among patients with first-episode psychosis. *J Psychiatr Res*. 2022;151:419–26.
23. McCarron MM, Schulze BW, Thompson GA, Conder MC, Goetz WA. Acute phencyclidine intoxication: incidence of clinical findings in 1,000 cases. *Ann Emerg Med*. 1981;10(5):237–42.
24. Pikwer A. Depersonalization disorder may be related to glutamate receptor activation imbalance. *Med Hypotheses*. 2011;77(4):593–4.
25. Mechri A, Saoud M, Khiari G, d’Amato T, Dalery J, Gaha L. Glutaminergic hypothesis of schizophrenia: clinical research studies with ketamine. *Encephale*. 2001;27(1):53–9.
26. Pomarol-Clotet E, Honey GD, Murray GK, Corlett PR, Absalom AR, Lee M, et al. Psychological effects of ketamine in healthy volunteers. *Phenomenological study*. *Br J Psychiatry*. 2006;189:173–9.
27. Varley J, Vincent A, Irani SR. Clinical and experimental studies of potentially pathogenic brain-directed autoantibodies: current knowledge and future directions. *J Neurol*. 2015;262(4):1081–95.
28. Miya K, Takahashi Y, Mori H. Anti-NMDAR autoimmune encephalitis. *Brain Dev*. 2014;36(8):645–52.
29. Funayama M, Mizushima J, Takata T, Koreki A, Mimura M. Altered perception might be a symptom of anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis. *Neurocase*. 2018;24(5–6):255–8.
30. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974;2(7872):81–4.
31. Ideno Y, Takayama M, Hayashi K, Takagi H, Sugai Y. Evaluation of a Japanese version of the Mini-mental State examination in elderly persons. *Geriatr Gerontol Int*. 2012;12(2):310–6.
32. Quinn TJ, Dawson J, Walters MR, Lees KR. Reliability of the modified Rankin Scale: a systematic review. *Stroke*. 2009;40(10):3393–5.
33. Shinohara Y, Minematsu K, Amano T, Ohashi Y. Modified Rankin scale with expanded guidance scheme and interview questionnaire: interrater agreement and reproducibility of assessment. *Cerebrovasc Dis*. 2006;21(4):271–8.
34. Allman MJ, Meck WH. Pathophysiological distortions in time perception and timed performance. *Brain*. 2012;135(Pt 3):656–77.
35. Teixeira S, Machado S, Paes F, Velasques B, Silva JG, Sanfem AL, et al. Time perception distortion in neuropsychiatric and neurological disorders. *CNS Neurol Disord Drug Targets*. 2013;12(5):567–82.
36. Stanghellini G, Ballerini M, Presenza S, Mancini M, Raballo A, Blasi S, et al. Psychopathology of Lived Time: abnormal time experience in persons with Schizophrenia. *Schizophr Bull*. 2016;42(1):45–55.
37. Fouks L, Guibert S, Montot M. Minkowski’s concept of lived time. *Ann Med Psychol (Paris)*. 1989;147(8):801–9.
38. Martin B, Franck N, Cermolacce M, Coull JT, Giersch A. Minimal self and timing disorders in Schizophrenia: a Case Report. *Front Hum Neurosci*. 2018;12:132.
39. Cutting J, Silzer H. Psychopathology of time in brain disease and schizophrenia. *Behav Neurol*. 1990;3(4):197–215.
40. Blom JD, Nanuashvili N, Waters F. Time distortions: a systematic review of cases characteristic of Alice in Wonderland Syndrome. *Front Psychiatry*. 2021;12:668633.
41. Blom JD. Alice in Wonderland syndrome: a systematic review. *Neurol Clin Pract*. 2016;6(3):259–70.
42. Mastroia G, Mancini V, Viganò A, Di Piero V. Alice in Wonderland Syndrome: a clinical and pathophysiological review. *Biomed Res Int*. 2016;2016:8243145.
43. Giannotti AM. Síndrome De Alicia en El País De Las Maravillas E infección por virus de Epstein Barr. *Arch. Argent. Pediatr*; 2003. pp. 41–3.
44. Pérez Méndez C, Martín Mardomingo M, Otero Martínez B, Lagunilla Herrero L, Fernández Zurita C. [Alice in Wonderland syndrome due to Epstein-Barr virus infection]. *Esp Pediatr*. 2001;54(6):601–2.
45. Augarten A, Aderka D. Alice in Wonderland syndrome in H1N1 influenza: case report. *Pediatr Emerg Care*. 2011;27(2):120.
46. Hansen N. NMDAR autoantibodies in psychiatric disease - an immunopsychiatric continuum and potential predisposition for disease pathogenesis. *J Transl Autoimmun*. 2022;5:100165.
47. Hansen N, Luedecke D, Maier HB, Neyazi A, Fitzner D, Wiltfang J, Malchow B. NMDAR1 autoantibodies as potential biomarkers for schizophrenia phenotyping. *Lancet Psychiatry*. 2024;11(10):780–1.

Publisher’s note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.