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Memantine-Assisted Treatment of N-Methyl-D-Aspartate Receptor Antibody Encephalitis: A Mini Review

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Abstract: N-methyl-D-aspartate receptor encephalitis (NMDARE) presents serious neurological manifestations such as reduced consciousness, seizures, and movement disorders, which can escalate to coma or severe autonomic dysfunction. Treatment typically involves immunotherapy and tumor removal to mitigate the autoimmune response. Timely diagnosis and treatment are critical to prevent severe neurological impairment or death. Memantine, an NMDA receptor antagonist, has shown variable effectiveness in treating NMDARE according to several case reports, yet comprehensive analyses remain scarce. This mini review draws on five literature sources and eight case studies from databases including PubMed, Embase, the Cochrane Library, and Web of Science, highlighting both the potential and risks of memantine as an adjunct therapy. We explore how memantine may reduce symptoms by blocking excessive NMDA receptor (NMDAR) antibody binding, while potentially worsening symptoms by reducing extracellular NMDAR availability, thus impairing neuronal communication. This dual effect calls for further investigation into the optimal use and duration of memantine treatment in NMDARE management.

Keywords: N-methyl-D-aspartate, N-methyl-D-aspartate receptor, N-methyl-D-aspartate receptor antibody, N-methyl-D-aspartate receptor antibody encephalitis, Memantine

Introduction

N-methyl-D-aspartate receptor encephalitis (NMDARE) is one of the most common forms of autoimmune encephalitis, associated with the immune system attacking N-methyl-D-aspartate receptors (NMDAR) in the brain.[1] It presents with a variety of clinical symptoms, which can be categorized into different stages. Early symptoms often mimic the flu, including fever and headache. Patients may then develop psychiatric symptoms such as hallucinations, abnormal behavior, and psychosis. Other symptoms include cognitive impairment disorder, speech difficulties, seizures, movement disorders, impaired consciousness, and autonomic dysfunction. In severe cases, patients can slip into a coma.[2] NMDARE is most likely to affect children and adolescents, particularly females. The acute phase is severe, with a mortality rate of approximately 5%, and about 15% of patients may experience relapses.[3,4] The 2021 International Consensus recommends early intervention, hormonal therapy, plasma exchange, immunosuppression, and symptomatic treatment to improve the prognosis and comorbidities of NMDARE and to reduce the risk of relapse.[5] However, the therapeutic mechanisms of NMDARE remain incompletely understood, and there is still a lack of consensus, particularly regarding the use of memantine, an NMDA antagonist, in treating this disease.

Memantine is an NMDA receptor antagonist that works by reducing the action of the excitatory neurotransmitter glutamate in the brain.[6] In NMDARE, the impaired NMDA receptors lead to an imbalance of neurotransmitters, resulting in neurological dysfunction. The potential mechanism of action of Memantine involves modulating this imbalance by decreasing the excessive activation of NMDA receptors, thereby potentially restoring neurological function, and

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improving the symptoms of cognitive impairment disorder of NMDARE.[7,8] In recent years, there have been numerous case reports on the role of Memantine in the treatment of NMDARE and its complications. However, the use of Memantine in treating this condition has shown a certain dichotomy in its effects, and there is a lack of systematic summarization and in-depth analysis of these cases. This suggests that more research is needed to clarify the efficacy and potential limitations of Memantine in treating NMDARE.

In this study, we conducted a mini review searching for case reports of memantine-assisted treatment of NMDARE to explore its intrinsic mechanism of action and clinical applications in NMDARE and its complications.

Method

Literature Search

The databases searched included PubMed, Embase, the Cochrane Library, Web of Science, China National Knowledge Infrastructure and China Wanfang Database. The search terms as follows: ("Memantine" [Mesh] OR "Memantine" OR "1,3-Dimethyl-5-aminoadamantane" OR "1-Amino-3,5-dimethyladamantane" OR "Namenda" OR "Ebixa" OR "Memantine Hydrochloride" OR "Axura" OR "D-145" OR "D 145" OR "D145") AND ("Anti-N-Methyl-D-Aspartate Receptor Encephalitis" [Mesh] OR "Anti N Methyl D Aspartate Receptor Encephalitis" OR "Anti-N-Methyl-D-Aspartate Receptor Encephalitides" OR "Encephalitides, Anti-N-Methyl-D-Aspartate Receptor" OR "Encephalitis, Anti-N-Methyl-D-Aspartate Receptor" OR "Anti-NMDA Receptor Encephalitis" OR "Anti NMDA Receptor Encephalitis" OR "Anti-NMDA Receptor Encephalitides" OR "Encephalitides, Anti-NMDA Receptor" OR "Encephalitis, Anti-NMDA Receptor" OR "Receptor Encephalitides, Anti-NMDA" OR "Receptor Encephalitis, Anti-NMDA" OR "Anti-NMDAR Encephalitis" OR "Anti NMDAR Encephalitis" OR "Anti-NMDAR Encephalitides" OR "Encephalitides, Anti-NMDAR" OR "Encephalitis, Anti-NMDAR" OR "Non-paraneoplastic Anti-N-Methyl-D-Aspartate Receptor Encephalitis" OR "Non paraneoplastic Anti N Methyl D Aspartate Receptor Encephalitis" OR "Non-paraneoplastic Anti-NMDA Receptor Encephalitis" OR "Non paraneoplastic Anti NMDA Receptor Encephalitis" OR "Non-paraneoplastic Anti-NMDAR Encephalitis" OR "Anti-NMDAR Encephalitides, Non-paraneoplastic" OR "Anti-NMDAR Encephalitis, Non-paraneoplastic" OR "Encephalitides, Non-paraneoplastic Anti-NMDAR" OR "Encephalitis, Non-paraneoplastic Anti-NMDAR" OR "Non paraneoplastic Anti NMDAR Encephalitis" OR "Non-paraneoplastic Anti-NMDAR Encephalitides" OR "Paraneoplastic Anti-N-Methyl-D-Aspartate Receptor Encephalitis" OR "Paraneoplastic Anti N Methyl D Aspartate Receptor Encephalitis" OR "Paraneoplastic Anti-NMDA Receptor Encephalitis" OR "Paraneoplastic Anti NMDA Receptor Encephalitis" OR "Paraneoplastic Anti NMDA Receptor Encephalitis" OR "Anti-NMDAR Encephalitides, Paraneoplastic" OR "Anti-NMDAR Encephalitis, Paraneoplastic" OR "Encephalitides, Paraneoplastic Anti-NMDAR" OR "Encephalitis, Paraneoplastic Anti-NMDAR" OR "Paraneoplastic Anti NMDAR Encephalitis" OR "Paraneoplastic Anti-NMDAR Encephalitides"). The search period spanned from January 2000 to April 22, 2024. Secondary searches were supplemented by references from the included articles. The search process was carried out by two researchers independently for the purpose of obtaining correct search results without bias and ensuring a comprehensive extraction of relevant case reports, aiming to synthesize new insights into the treatment of NMDARE with memantine (Figure 1).

Inclusion and Exclusion Criteria

In the study, the inclusion criteria were cases formally diagnosed with NMDARE. The exclusion criteria were as follows: duplicate case reports, extended reports of the same case in favor of the most comprehensive and recent report, unconfirmed case reports, and cases lacking essential clinical details.

Data Extraction

The data retrieved from the articles included: 1) Basic information about the study, such as the year of publication, the name of the first author, the location of the study, and the number of participants; 2) Basic information about the patients, including gender, age, diagnosis, medication, and prognosis.

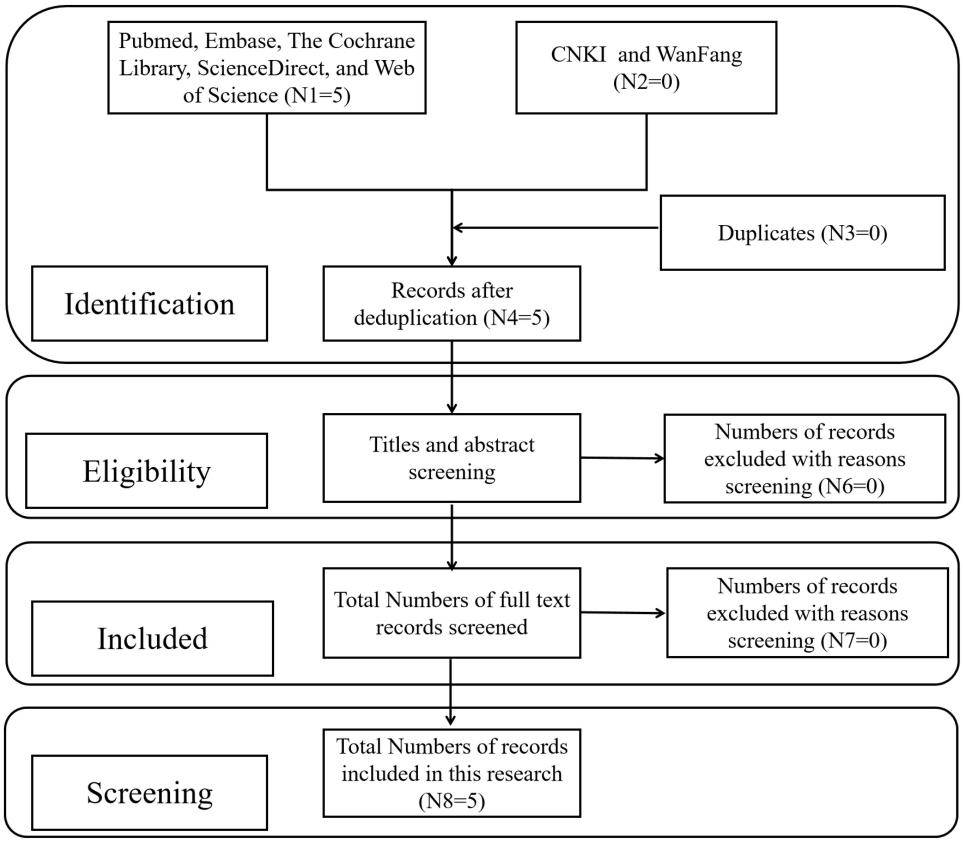

Figure 1 Literature Search Flowchart.
Abbreviation: CNKI, China National Knowledge Infrastructure.

Result

A total of 5 articles and 8 cases were retrieved from PubMed, Embase, The Cochrane Library, and Web of Science databases (Tables 1 and 2). These cases involved 6 female and 2 male patients, with an average age of 26.1 years, originating from Taiwan, China (1 case), the USA (6 cases), and Mexico (1 case).[9–13] Seven of the cases tested positive for NMDAR antibodies, confirming a diagnosis of NMDAR encephalitis. Among the confirmed cases, one was associated with an ovarian teratoma, one with a testicular tumor; four cases showed no tumor presence, and two lacked tumor information. Steroids and IVIG were selected as the preferred first-line immunotherapy regimen in all cases. In refractory cases, plasma exchange, rituximab, and cyclophosphamide were considered as second-line therapies. After adding memantine treatment, five patients showed complete resolution of clinical symptoms, with their BFCRS (Bush-

Table 1 Basic Information About the 7 Patients

No.	Genders	Age	Antibody Titer	Secondary Disease	Tumor Presence	Country or Area
1	Woman	21	Not mentioned	Hypoventilation, altered consciousness, sensory impairment, and memory impairment	No	China, Taiwan[9]
2	Woman	31	Positive	Catatonia	Not mentioned	New York, USA[10]
3	Woman	20	Positive	Catatonia	Ovarian teratoma	New York, USA[10]
4	Woman	26	Positive	Catatonia and seizure-like activity	No	New York, USA[10]
5	Woman	44	Positive	Catatonia	Not mentioned	Mexico[11]
6	Man	25	Positive	Catatonia	Testicular tumor	State of Texas, USA[12]
7	Woman	25	Positive	Catatonia and seizure	No	State of Texas, USA[12]
8	Man	17	Positive	Catatonia and seizure	No	State of Texas, USA[13]

Table 2 Effect of Memantine in Combination with Other Treatment Modalities in Patients with NDARE

No.	Baseline Treatment	Other Drugs	Glutamate Antagonist	Duration of Treatment	Relief
1	Hormonal Pulse, Plasma Exchange	No	Memantine	1 month	Yes
2	Steroids, IVIG, rituximab, plasmapheresis, and cyclophosphamide	Lorazepam 2 mg, thrice daily, causing respiratory depression	Amantadine capsules 100 mg and Memantine 10 mg.	Stopped due to agitation	Worsen
3	IVIG, Methylprednisolone, Rituximab, Ovarian Teratoma Resection	Limited efficacy of Lorazepam	Memantine 5–10 mg twice daily	2 months	Yes
4	IVIG, steroids, Lorazepam, levetiracetam, rituximab	Limited efficacy of Lorazepam	Memantine 5–10 mg daily	2 weeks	Yes
5	Olanzapine, Quetiapine, Methylprednisolone, Immunoglobulin Therapy	Limited efficacy of Lorazepam and Dexmedetomidine	Memantine 10–20 mg and Levetiracetam	1 year	Yes
6	IVIG, Methylprednisolone, Testicular tumor Resection	Limited efficacy of Lorazepam, valproic acid IV and aripiprazole	Memantine 5–10 mg twice daily	1 year	Yes
7	IVIG, Methylprednisolone, rituximab, and cyclophosphamide	Limited efficacy of Lorazepam, valproic acid IV and electroconvul-sive therapy	Memantine 10 mg twice daily	Not mentioned	BFCRS dropped to 6 and was discharged
8	IVIG, Methylprednisolone, Rituximab, Lorazepam	Acyclovir, Doxycycline, Levetiracetam, Phenytoin, Phenobarbital, Haloperidol, Olanzapine, Quetiapine	Memantine	Not mentioned	Worsen

Abbreviations: IVIG, Intravenous immunoglobulin; BFCRS, Bush-Francis Catatonia Rating Scale.

Francis Catatonia Rating Scale) scores dropping to 0. One patient, due to financial reasons, did not receive the fully optimized treatment and was discharged with a BFCRS score of 6. One patient did not respond to memantine treatment. One patient stopped taking the drug because she was too agitated.

Discussion

NMDARE is a commonly observed autoimmune encephalitis.[14] Its clinical manifestations primarily include headache and fever, followed by psychiatric disorders, behavioral abnormalities, cognitive dysfunction, impaired proximal memory, language dysfunction, and movement disorders. These are often accompanied by symptomatic epilepsy and catatonia.[5] Catatonia, one of the manifestations, is characterized by a range of psychomotor and volitional disorders. Its clinical manifestations encompass stereotypy, posturing, rigidity, movement mimicry, mutism, defiance, automatisms, and impulsive behaviors, among others.[15,16]

NMDA, a naturally occurring amino acid derivative in animals, is an important excitatory neurotransmitter and a homologue of L-glutamate in the mammalian central nervous system.[17] NMDA is an agonist of the NMDAR. While glutamate is the primary neurotransmitter acting on the NMDAR, NMDA can substitute for glutamate to some extent.[18] However, NMDA uniquely binds only to disease-regulated NMDARs and does not affect other types of glutamate receptors. NMDA plays a role in regulating the hypothalamic-pituitary growth axis. An optimal level of NMDA promotes the secretion of somatostatin in hypothalamic neuronal cells and increases intracellular calcium ion levels.[19] Excessive activity of NMDA leads to agonism and can result in seizures.[20] NMDAR, a subtype of excitatory glutamate receptors, function at synapses—the junctions between neurons—to regulate neuronal communication by receiving neurotransmitter signals.[20,21] Overactivation of NMDAR during seizures can cause ionic imbalance across the cell membrane. This activates neurotoxic signaling pathways, leading to neuronal damage, apoptosis, neurological dysfunction, and ultimately, cognitive impairment.[21] Autoimmune dysfunction leads to the production of antibodies against the NMDAR, targeting it for attack.[5] These antibodies cause a reversible internalization of the NMDAR, leading to its

hypofunction and an extracellular glutamate overload. This impacts the limbic system and triggers a widespread immune response in the brain, culminating in the development of NMDARE.[22]

In clinical practice, when catatonia is evident in patients with NMDARE, initial treatment typically involves administering a stimulating dose of lorazepam, which is gradually increased based on the patient's response. If the patient does not respond adequately to lorazepam or when electroconvulsive therapy (ECT) is unavailable or contraindicated, memantine is often introduced as a supplemental treatment, especially when malignant catatonia is suspected. In addition to lorazepam and memantine, a study involving 41 NMDARE patients showed that other medications, such as bromocriptine, amantadine, and L-dopa, were also used to treat the catatonia in NMDARE patients. Although these drugs are not sufficient to completely relieve catatonia symptoms, they can help slow the progression of catatonia and control psychomotor agitation.[23]

Studies on memantine's role in NMDARE treatment have produced mixed results. A literature review showed that memantine was effective in treating one case of NMDARE during immunosuppressive therapy, hormonal therapy, or plasma exchange. Additionally, it was effective in 5 out of 7 cases of post-NMDARE catatonia, although in the remaining 2 cases, memantine worsened the condition. These findings highlight that memantine's impact on NMDARE-related catatonia can be variable: while it may offer an adjuvant therapeutic effect in some cases, it could also increase the risk of clinical deterioration in others.

Previous studies have suggested that memantine, when combined with neuroprotective agents and immunotherapy, can alleviate acute-phase symptoms of severe NMDARE.[9] However, the potential influence of other variables remains unclear, and some researchers have raised concerns that memantine's action on NMDAR could exacerbate NMDARE symptoms.[11] Further analysis of treatment strategies suggests that memantine may be considered when first- and second-line treatments fail to relieve catatonic symptoms, but careful evaluation of its timing and dosage is essential. Typically, the initial dose of memantine is 5 mg twice daily, with the potential to increase to 10 mg twice daily to minimize the risk of symptom exacerbation or adverse effects. The duration of memantine treatment can vary, ranging from 2 weeks to 1 year, depending on the patient's response.

To address these complex issues related to treatment strategies, this discussion synthesizes conclusions drawn from our rigorous data extraction process, which carefully considered individual characteristics and treatment histories of each case. We propose a dual mechanism for memantine, as an NMDA antagonist, in the treatment of NMDARE. During the acute phase of NMDARE, NMDAR antibodies induce receptor internalization, leading to disruptions in receptor function and elevated levels of extracellular glutamate, which may result in excitotoxicity. As first- and second-line treatments help restore NMDAR function, the excessive glutamate produced during the acute phase can overstimulate the receptor, exacerbating catatonic symptoms. In this context, memantine may serve to modulate this excessive glutamatergic activity by blocking the overactivation of NMDARs at the ion channel level, thereby helping to alleviate catatonic episodes and improve clinical outcomes. While memantine's action within the receptor channel helps manage glutamate excitotoxicity, it does not directly interfere with antibody binding at the extracellular domain of NMDARs. Instead, it primarily regulates the receptor's ion channel function, preventing overstimulation by glutamate and restoring more balanced neuronal communication. However, in cases where NMDAR hypofunction is more pronounced due to antibody-induced receptor internalization, further administration of memantine could potentially reduce already limited receptor activity, which might worsen some symptoms (Figure 2). These findings underscore the importance of tailoring memantine use to the specific clinical context of each NMDARE patient and highlight the need for additional studies to refine the treatment strategies and dosing protocols for managing catatonia and related symptoms in NMDARE.

In this study, we identified only a small number of cases (8) through the screening database, which limits the generalizability of our findings. Additionally, there were variations in the treatment regimens among the reviewed cases. This heterogeneity may complicate the assessment of memantine's effect on disease outcomes. Each case's unique context, including the presence of confounding therapies, significantly influences the treatment outcomes and our understanding of memantine's role in NMDARE treatment.

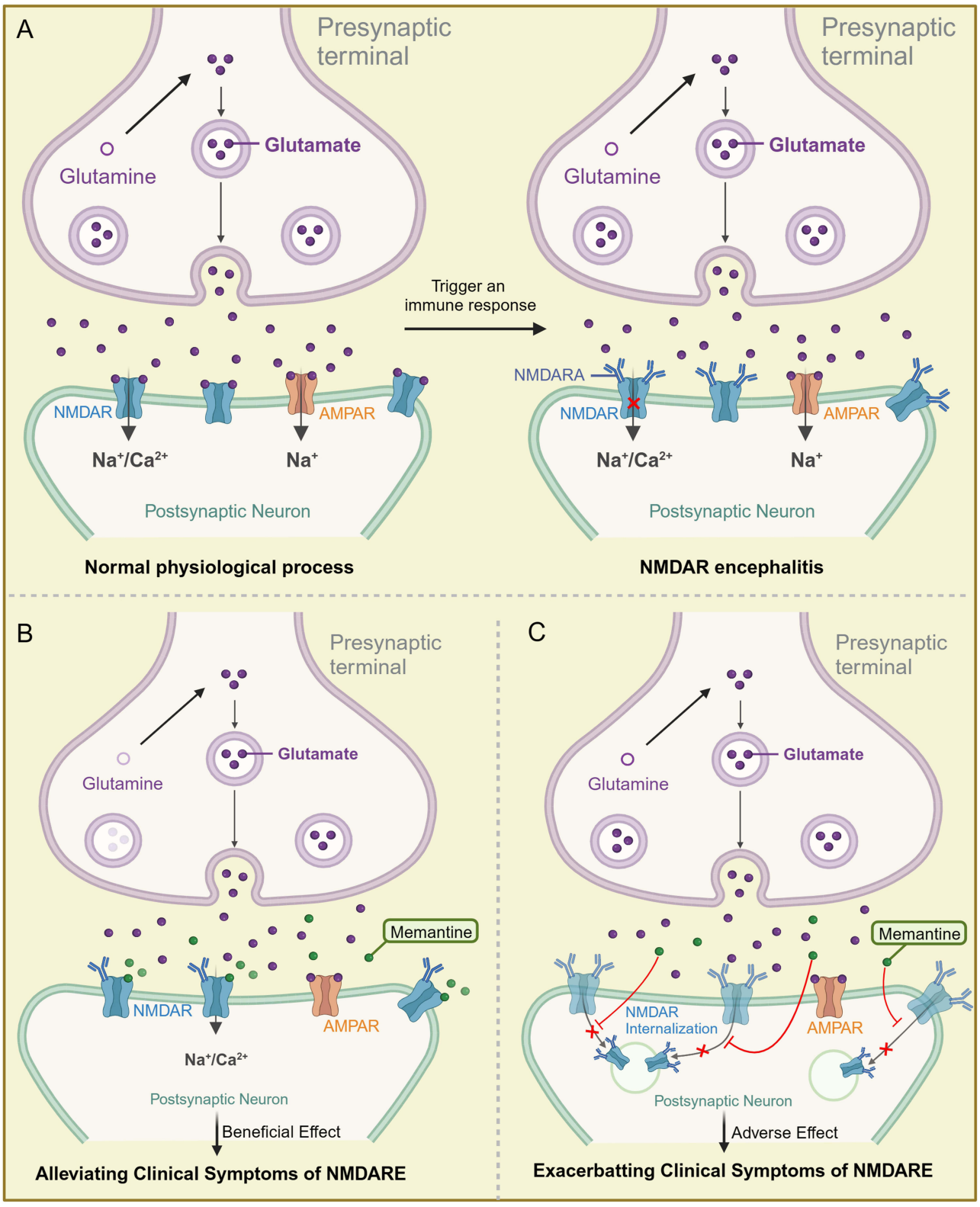

Figure 2 Potential mechanism of action for memantine in competitively binding NMDAR with NMDAR Antibodies. (**A**) Under physiological conditions, glutamate in the CNS binds to NMDARs and AMPARs on postsynaptic neurons. When there is a disturbance in the immune system, antibodies are produced against NMDA receptors in the brain, triggering NMDAR encephalitis; (**B**) In the early stage of NMDAR antibody binding to NMDARs, memantine can compete with the NMDAR antibody for NMDAR binding and prevent excessive antibody attachment to NMDARs; (**C**) Memantine inhibits the reversible internalization of NMDARs induced by NMDAR antibodies, thereby blocking NMDAR activity and impairing neuronal communication. Created in BioRender. Liu, F. (2024) https://BioRender.com/c70p190.

Abbreviations: NMDAR, N-Methyl-D-Aspartate receptor; AMPAR, α-Amino-3-Hydroxy-5-Methyl-4-Isoxazolepropionic acid receptor; NMDARE, N-Methyl-D-Aspartate Receptor Encephalitis.

Conclusion

In summary, this literature review indicates that NMDARE, an autoimmune encephalitis linked to NMDAR antibodies, frequently results in neurological and psychiatric symptoms. Our analysis reveals the complexity of memantine's effects on NMDARE, emphasizing the critical need for personalized treatment approaches and further research to refine therapeutic strategies for this challenging condition.

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Disclosure

The authors report no conflicts of interest in this work.

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