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LETTER TO THE EDITOR

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Effective use of memantine for catatonia in major depressive disorder after failure of electroconvulsive therapy: A case report

Catatonia is a neuropsychiatric disease characterized by abnormal psychomotor signs, such as immobility or excessive motor activity. The diagnosis is confirmed when at least three of the following symptoms are observed: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation, grimacing, echolalia, or echopraxia.[1] According to a meta-analysis of 74 studies, the prevalence of catatonia was approximately 9% and it is often comorbid with psychiatric disorders and medical illness.[2] While benzodiazepine and electroconvulsive therapy (ECT) are standard treatments for catatonia,[3–5] approximately 30% of patients with catatonia fail to improve.[5] A recent systematic review has referred to glutamate receptor antagonist medications as the option following benzodiazepine medication and ECT.[3] In this case report, we describe a major depressive disorder (MDD) patient with treatment-resistant catatonia who did not respond to ECT and benzodiazepine medication; the symptoms were improved on the administration of memantine, a glutamate receptor antagonist.

A 54-year-old male patient presented with a medical history of Guillain–Barre syndrome and myocardial infarction. At age 50, he had had insomnia due to stress from work and family problems and was prescribed a hypnotic at a nearby clinic. At age 52, his depressive symptoms had gradually worsened. He was diagnosed with MDD and prescribed mirtazapine 15 mg/day, which improved his symptoms. At age 53, his depressive symptoms had relapsed, which retarded his thinking and interfered with his daily life; he was admitted to a psychiatric hospital. After that, he additionally presented with stupor, catalepsy, mutism, negativism, and agitation. The blood samples and head computed tomography (CT) scans showed no abnormal findings. Following an intravenous injection of 10 mg diazepam, his symptoms partially improved, and he was diagnosed with catatonia. Lorazepam was administered at a dose of 8 mg/day; however, it did not lead to remission. Additionally, the creatinine kinase (CK) levels elevated to 1100 U/L, and the body temperature rose to 37.5°C; the medical team decided to perform ECT. After the eighth ECT session, the patient presented with impaired cognitive function and failure to achieve remission. The patient was transferred to our hospital.

As the patient had been in a stupor and could not take oral intake, a nasogastric tube was inserted. In addition to quetiapine at 200 mg/day that had been previously prescribed, we started lorazepam at 3 mg/day and increased that to 27 mg/day (by 1–2 mg/day, while assessing the patient's response to the treatment at intervals of 2–3 days). We ruled out other medical problems with the help of a neurologist's consultation as well as blood tests, cerebrospinal fluid tests, electroencephalogram, CT, and magnetic resonance imaging (MRI), beginning the administration of memantine because of the poor efficacy of lorazepam in catatonia. After increasing the memantine dose to 20 mg, the agitation improved, and the patient could speak with us. However, the eosinophil levels elevated, and the patient complained of depressive symptoms; amitriptyline at 30 mg/day was initiated. Memantine was discontinued due to elevated eosinophils as a suspected side-effect, which in turn led to the exacerbation of agitation. We decreased the dosages of amitriptyline and quetiapine, and increased the olanzapine dosage to manage the increase in agitation. Lorazepam dosages were reduced because high doses of it did not achieve remission, and we found that memantine did not increase eosinophil and decided to re-administer it because increased eosinophil had been lasting after discontinuation of administration. Consequently, the agitation and stupor gradually improved, and the patient was able to eat, walk by himself, and speak. Finally, he was discharged on the 186th day from admission (the course of catatonia symptoms and treatment is shown in Figure 1).

To our best knowledge, this is the first case report of memantine improving the symptoms of an MDD patient with treatment-resistant catatonia who did not respond to ECT and high doses of benzodiazepine medication without any sequelae. A previous case report revealed that ECT could not improve the symptoms of an MDD patient with catatonia, and was effectively improved by amantadine, which is a glutamate receptor antagonist medication, like memantine.[6] We suggest that glutamate receptor antagonists

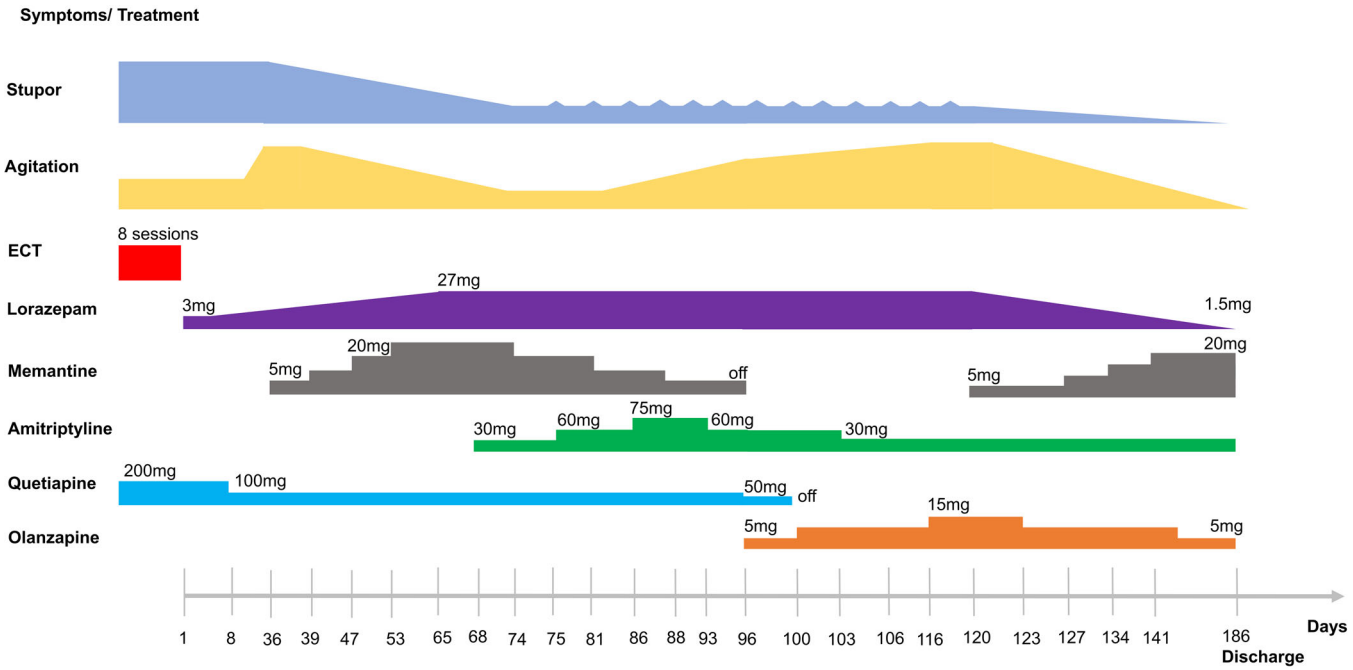

FIGURE 1 The courses of catatonia symptoms and treatment. ECT, electroconvulsive therapy.

may be an effective medication for MDD patients with ECT and benzodiazepine-resistant catatonia.

AUTHOR CONTRIBUTIONS

Akitoyo Hishimoto designed the research procedure. Takaki Tanifuji was responsible for drafting of the manuscript. Takaki Tanifuji, Ikuo Otsuka, Toshio Atarashiya, Atsushi Kimura, Tadasu Horai, and Satoshi Okazaki collected the data. Ikuo Otsuka mainly revised the manuscript. All authors read and approved the final submission.

CONFLICT OF INTEREST STATEMENT

N/A

DATA AVAILABILITY STATEMENT

Requests to access these datasets should be directed to the corresponding author.

ETHICS APPROVAL STATEMENT

We implemented this study design and all related procedures in accordance with the Declaration of Helsinki. This study was not approved by the Ethical Committee because it is a case report. We explained the off-label use of memantine to the patient and family and obtained their consent.

PATIENT CONSENT STATEMENT

The patient provided informed consent to receive treatment and for the publication of this case report. We gave consideration to preserve the anonymity of the patient.

CLINICAL TRIAL REGISTRATION

N/A

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