Successful Treatment of Treatment-Resistant Schizophrenia in a 10-Year-Catatonic Patient by Augmentation of Selective Serotonin Reuptake Inhibitors

A Case Report

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Abstract: Although catatonia is a well defined syndrome, the treatment of chronic catatonia remains an unresolved issue. Here, we report a successful treatment of a 30-year-old patient with treatment-resistant catatonic schizophrenia in 10 years by augmentation of selective serotonin reuptake inhibitors (SSRIs).

We present a 30-year-old man with treatment-resistant catatonic schizophrenia who failed to respond to the treatment of benzodiazepines and antipsychotics for 10 years. He markedly improved after taking SSRIs. Now, he does not hold odd postures and begins to talk and show more facial expressions.

We postulate that the therapeutic effect is related to the enhancement of 5-HT neurotransmission. SSRIs can be a considerable choice to treat chronic catatonia.

INTRODUCTION

Catatonia is characterized by immobility, mutism, negativism, and peculiar motor behavior. Catatonia may be caused by physical or psychiatric disorders. It has been described in different psychiatric conditions including schizophrenia and affective disorders. Approximately 10% to 15% of patients with catatonia meet the criteria for schizophrenia. Although there are some new therapeutics being developed to treat catatonia, such as N-methyl-D-aspartate receptor antagonists, antipsychotics, repetitive transcranial magnetic stimulation, benzodiazepines, and electroconvulsive therapy (ECT) are still the mainstay treatments.

Previous studies indicated that different psychiatric diseases with catatonia responded differently to benzodiazepines. Catatonic patients with affective disorder show some response to benzodiazepines. Compared to patients with affective disorder, those with schizophrenia may have worse response to benzodiazepines. A difference in response to benzodiazepines also existed between acute and chronic catatonia. To treat chronic catatonia effectively remains an unresolved issue until now.

Selective serotonin reuptake inhibitors (SSRIs) are used to treat obsessive–compulsive disorders, panic disorders, eating disorders, and depression. Many studies have examined the effectiveness of antidepressants in treating psychiatric conditions other than depression. A meta-analysis showed that SSRIs may be effective for negative symptoms of schizophrenia. The aforementioned studies found that SSRIs can be widely applied to treat many other psychiatric disorders. We present a patient with chronic catatonic schizophrenia who failed to respond to the treatment with benzodiazepines and antipsychotics, but he markedly improved after taking SSRIs.

CASE REPORT

The patient is a 30-year-old man. He had auditory hallucinations and delusions of persecution and reference since senior high school. He gradually entered a catatonic state, which included catalepsy, mutism, posturing, mannerism, agitation, and grimacing. He had no apparent extrapyramidal symptoms after taking antipsychotics. Due to chronic psychosis with marked deterioration of psychosocial function, he has been admitted to our psychiatric day ward for rehabilitation since he was 19-years old. His laboratory examinations and brain image study showed no obvious abnormality. The catatonic-type schizophrenia was diagnosed according to The Diagnostic and Statistical Manual of Mental Disorders (4th Ed; DSM-IV) criteria. His younger brother had a diagnosis of schizophrenia. He was changed from sulpiride to haloperidol and lithium carbonate 600 mg/day for 7 years. His positive response to augmentation of SSRIs is described as follows.
symptoms mildly improved, but the catatonic symptoms persisted. He maintained odd postures, such as half-squatting while gazing at the floor or holding one hand up horizontally in front of the mirror for around 2 hours every day. He became skinny because of poor nutrition. We added fluoxetine 40 mg/day for him. Two weeks later, some interruptive behavior occurred. Fluoxetine was replaced directly by paroxetine, which has more sedative effect (paroxetine 40 mg/day). After another 2 weeks, catatonic symptoms dramatically improved.

We kept his regimen of medications for the next 19 months. The Positive and Negative Syndrome Scale improved from 148 to 106. There was partial remission of psychotic symptoms but no relapse of catatonic symptoms. He does not hold odd postures anymore. He is able to interpret the contents of the voice hallucination that he had experienced and described a female voice that commanded him to maintain a certain posture. In addition, he began to show more facial expressions and even chat with hospital staff and his family.

DISCUSSION

The present case is characterized by treatment-resistant to antipsychotics and catatonic symptoms with insufficient response to benzodiazepines. Surprisingly his symptoms markedly improved after using SSRIs. According to Huang study, more than two-thirds of catatonic patients responded to high doses of lorazepam. However, in our case, using benzodiazepine, haloperidol, and lithium carbonate, his psychotic and catatonic symptoms did not improve. Some studies indicated that benzodiazepines were effective in the treatment of acute catatonia, but were less effective in patients with chronic catatonic schizophrenia. According to the review article, there is one case report showed that the use of fluoxetine might be useful in treating catatonic symptoms. Consoli et al reported that a catatonic patient, who had a moderate response to lorazepam treatment, improved after using fluoxetine for 40 days. A study about baclofen-induced catatonia rats found that enhanced 5-HT neurotransmission significantly reversed baclofen-induced catatonia via 5-HT uptake inhibitor (eg, fluoxetine) or 5-HT releaser. In contrast, 5-HT1A antagonist (eg, Pindolol) and 5-HT2 antagonist (eg, sulpiride), which inhibit the 5-HT neurotransmission, exacerbated catatonia. It appears that the manipulations of the central serotonergic mechanisms may modulate catatonia.

In this case, SSRI augmentation improves the catatonia and then his negative symptoms improved subsequently. It may be due to his catatonic symptoms were severe and would inhibit him to interact with others. However, it needs further investigation to clarify the mechanism.

We replaced fluoxetine by paroxetine. The two SSRIs have similar pharmacologic property except paroxetine with anti-mascarinic effect. Previous studies indicate that fluoxetine has efficacy in the treatment of catatonia. Our report is the first one to suggest that using paroxetine may also have efficacy in the treatment of catatonia. Psychiatrists may consider using paroxetine augmentation to treat catatonia in schizophrenia patients, because its sedative benefit would decrease interruptive behavior during recovering from catatonia.

This case implies that there are other possible treatments to catatonic schizophrenia. Further studies for mechanism of catatonia treatment are needed.

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