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Letter to the Editor

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A case study on attenuated forms of catatonic symptoms in schizophrenia: relevance of early intervention with electroconvulsive therapy

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To the Editor:

Catatonia is a well-known entity in psychiatry, which is predominantly a syndrome of motor dysregulation.[1] The prevalence of catatonia among patients with psychiatric illness varies. Catatonic symptoms are seen beyond schizophrenia and have wide varieties of presentation.[2] Several medical disorders, including neurological disorders, present with symptoms of catatonia too. The pathophysiology of catatonia is complex and mainly involves neurotransmitters like GABA and glutamate.[3] The treatment of catatonia is primarily dependent on benzodiazepines or electroconvulsive therapy (ECT).[2,4] Medications are the first-line treatment in the management of catatonia. It is often difficult to predict:

- *Which individual with catatonia will respond to benzodiazepines?*
- *Which catatonic symptoms will respond to benzodiazepines?*
- *What will be the dose of benzodiazepine, which will resolve the catatonic symptoms?*

Due to the lack of consensus and robust evidence, much time is wasted with experimentation with benzodiazepines in the management of catatonia. We here present a case of catatonic schizophrenia, where mutism was the prominent feature, and early intervention with ECT resulted in rapid resolution of the catatonic symptoms.

A 24-year-old male of average body built was admitted to Psychiatry with chief complaints of suspiciousness, fearfulness, increased irritability, increased physical activity, and reduced sleep, which in 6 months progressed to a state characterized by decreased oral intake and decreased verbal output, resulting in mutism. It persisted for one and half years. The onset of the complaints, as mentioned earlier, was insidious, and fluctuating in course. No precipitating factor could not be elicited for the symptoms. For his illness, he has been on medications (olanzapine up to 10 mg/day for at least 8 weeks and lorazepam up to 8 mg/day for 4 weeks) before his current psychiatric consultation, but there was no improvement in his verbal output. Due to nonresponse to antipsychotic medications and benzodiazepine, he was hospitalized for proper evaluation and management.

As per the available history, after few weeks of the onset of his psychiatric illness, psychiatric consultation was sought, and he was started with olanzapine 10 mg/day. On this medication, his suspiciousness, irritability, and sleep improved. Gradually, his family members began to notice that his verbal output got decreased. Before the illness, he was a cheerful person and was interactive, but he only replied in yes or no, and gradually, he stopped speaking and would communicate with hand gestures. His oral intake also decreased significantly. He would eat slowly. He also stopped taking solid meals and would only take fluids. There is no history of any difficulty swallowing, regurgitation, or holding of food or saliva in the mouth for a prolonged period. For these complaints, he was again taken to a private practitioner, and Tab. Clozapine was added along with Tab. Olanzapine 10 mg/day. Clozapine was started from 25 mg/day and increased to 75 mg/day. On these medications, his oral intake improved, and he started having solid meals but showed no improvement in verbal output. He was on these medications for 10 months, but after stopping these medications for 2 months, his symptoms reappeared, and later on, resuming these medications, again, he improved except for his inability to speak. In a follow-up visit, the psychiatrist stopped clozapine and added amisulpride 200 mg/day to the ongoing treatment regimen, but there was no further improvement. Family members took a consultation from an otorhinolaryngologist for his inability to speak, but no abnormality was detected on examination. Computed tomography brain was also done, which was within normal limits.

At the time of hospitalization, his Bush Francis Catatonia Rating Scale (BFCRS) score was 13. He was given parenteral (intravenous) lorazepam 8 mg over 24 hours in two divided doses, which resulted in a decrease in rigidity, but mutism persisted (the BFCRS score reduced to 10). His neurological examination was within normal limits, and blood investigations were also within normal limits. A diagnosis of catatonic schizophrenia was made as per the ICD-10 diagnostic criteria. With the consent of the family members, he was planned for ECT on the third day of

hospitalization. Following the first session of ECT, he showed improvement in his verbal output. He started speaking few words and interacting. After the second session of ECT, his catatonic symptoms resolved completely. A total of four ECTs were given. He tolerated well to ECT, and no side effects were reported. The patient's catatonic symptoms were present between March 2020 and August 2021, during which the COVID-19 pandemic badly hit the world. On the resolution of his catatonic symptoms, when he was asked about important developments and pandemic, he was able to narrate it clearly and consistently. The patient had also stated that he feared that whenever he would speak, his tongue would fall out, and hence, he did not speak for years. After 2 weeks of hospitalization, the patient was discharged with olanzapine 10 mg/day and lorazepam 4 mg/day in divided doses. At 4 weeks follow-up, the patient did not have catatonic symptoms; however, he reported excessive sedation and suspiciousness against people. The dose of olanzapine was increased to 15 mg/day, and lorazepam was reduced to 2 mg/day.

The above case has several unique features. First, the patient had a long duration of mutism as a prominent catatonic symptom. Trial with therapeutic doses of antipsychotics, like olanzapine (10 mg/d), amisulpride (200 mg/day), add on clozapine (up to 75 mg/day), lorazepam 4 mg /day (for at least 4 weeks), and intravenous trial of lorazepam 8 mg within 24 hours, did not produce any noticeable clinical benefits. However, a substantial improvement in catatonic mutism occurred following the first session of ECT and complete resolution of catatonic symptoms after the second session of ECT.

This indicates that the presence of specific indicators like long duration of illness, poor response to antipsychotic or initial doses of benzodiazepine may be considered for ECT. In this case, the patient had consulted our center recently, where an ECT facility is available, and was considered in the early phase of evaluation considering potential factors of nonresponse. Therefore, neuromodulation techniques like ECT may be considered in such patients as an early intervention strategy.

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