

# Rapid Response of Long-Standing, Treatment-Resistant Non-Catatonic Mutism in Paranoid Schizophrenia with Single ECT session

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## Abstract

**Context:** Mutism is a common manifestation of catatonia, but mutism due to other forms of psychopathology and neurological disorders have also been described. Although not common, long-standing mutism has also been a feature of non-catatonic schizophrenia and traditionally responds less to conventional therapies. **Case Report:** We describe a rare case of paranoid schizophrenia presenting with continuous mutism for about 4 years. This 26-year-old male had symptoms of schizophrenia without catatonia. After failed trial of adequate pharmacotherapy and psychological intervention and considering his level of dysfunction, he was started on electroconvulsive therapy (ECT). To our surprise, he improved with a single session of ECT while he was on concurrent pharmacotherapy. We also discuss the possible explanation for this rapid effect of ECT in such clinical presentation. To our knowledge, this is the first case of non-catatonic mutism of schizophrenia of this long duration responding so promptly to ECT, although there are other reports as well in literature, but multiple ECT sessions were applied in those cases. **Conclusion:** Non-catatonic mutism is perhaps presenting as a cultural variant in this part of the world and whenever encountered, ECT should be an option. Further research should be carried out to validate this idea.

**Keywords:** Electroconvulsive therapy, Mutism, Paranoid, Schizophrenia

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## Introduction

Mutism, though a manifestation of catatonic schizophrenia, has also been described in other forms of non-catatonic psychopathologies, although only a few of them described in literature.<sup>[1]</sup> In a study from Micronesian islands, 19 of 22 schizophrenic patients were having non-catatonic mutism in the initial phase, one among them for more than 20 years. Many of them responded to neuroleptics,<sup>[2]</sup> and there are also case reports of prolonged mutism in non-catatonic schizophrenia in the adult population.<sup>[3,4]</sup> Basu *et al.* and Grover *et al.* have separately reported the response

of long-standing non-catatonic mutism in paranoid schizophrenia to a combination of electroconvulsive therapy (ECT) and neuroleptics.<sup>[5,6]</sup>

We describe a case of non-catatonic mutism of 4 years, secondary to first-rank symptoms, which was refractory to antipsychotic treatment alone and responded to a single ECT session and was maintained on neuroleptics.

## Case Presentation

A twenty six year old unmarried man from rural area was brought to psychiatric emergency in January 2013 with acutely disruptive and violent behavior. The resident in-charge admitted the patient and on further interview found that that he had also stopped talking for past 4 years and communicated occasionally only through gestures and writing.

Upon further interview, his family members revealed that he was having premorbidly schizoid traits and his

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illness had started insidiously 8 years back, when he had just passed his secondary school and it progressed gradually. He had gradually deteriorated in academics, and self-care and his biological functions got disturbed. He left studies and started remaining aloof and dirty. He began refusing food prepared by his family and started cooking for himself, and also refused medications if prescribed. As could be noticed, he had developed delusions of persecution against his family members and neighbors, with second and third person hallucinations. He also had developed delusions of reference and would often quarrel with the people nearby.

For past 4 years, patient had stopped talking at all with further deterioration in other symptoms. He started communicating only through gestures and writing. These gestures will be shown only when necessary and were usually goal directed and purposeful. He did not give any reason for his mutism while writing down. He had been given medications like olanzapine 20 mg, quetiapine 700 mg, haloperidol 20 mg, clonazepam 4 mg, and other psychotropic agents, but the mutism did not improve. With passing time, the family did not pay much attention to his mutism and he remained in this condition for about 4 years. From past 2 years, he is living in an old abandoned house near his family. He was brought to our hospital only when he developed agitation and aggression and started creating violence at home.

On admission, the patient was shabby, unkempt, with long dirty beard and hairs, with poor eye contact, poor hygiene and self-whispering (lip movements only) although he could cough. There were no abnormal body movements, mannerism, rigidity, posturing, echolalia or echopraxia or any abnormality on ear, nose and throat (ENT) evaluation. His Positive and Negative Syndrome Scale (PANSS) score was calculated as 114 and Global Assessment of Functioning (GAF) score of 10-15. He was started on injection olanzapine 10 mg once daily and injection lorazepam 4 mg twice daily and a gap of more than 2 hours was ensured between the injectables. He was shifted to oral medication after 3 days, when his condition began to improve and he started accepting oral medication. After 1 week, patient showed a considerable improvement in psychotic features and self-care (PANSS 86) with a GAF score of 15-20. Olanzapine was increased to 20 mg. He continued to improve in psychotic features for next 3 weeks but continued to be mute (PANSS 70, GAF 15-20) while as Bush-Francis Catatonia Rating Scale (screening) score was 1, thus ruling out catatonia. His baseline investigations including hemogram, liver and Kidney profile, blood sugar, electrolytes and metabolic profile were normal. His urinary drug screen was negative. Magnetic resonance imaging (MRI) of brain and electroencephalogram were reported to be normal, and there was no neuro-deficit, or cognitive decline (Mini

Mental State Examination [MMSE] = 27/30) or any other abnormality on systemic examination that could explain the mutism. At 4 weeks, his PANSS was 62, but the mutism did not go. In the mean time, multiple interventions were tried including psychological intervention, suggestions, and interview under the effect of lorazepam (multiple times). However, his mutism failed to respond to these interventions. Patient was discussed in clinical meeting designated for "difficult and unusual cases" with whole unit including chief consultant. A trial of ECT sessions was decided for mutism, in addition to usual medication and care. Attendants gave written consent for the same after a ward resident educated them about benefits and side effects of such treatment.

A session of modified bilateral ECT was administered using a brief-pulse constant energy machine. Glycopyrrolate, ondansetron, and ranitidine were used as pre-medications, and propofol (90 mg) was the inducing agent. Succinyl-choline was the relaxing agent. During the procedure, the duration of current was 2 seconds, at frequency of 70 Hz and the pulse width of 1 ms. The seizure duration was 28 seconds (motor seizure). The next ECT was to be delivered 2 days later, but to our surprise, the patient started talking monosyllables 3 hours after the ECT session and as the day passed by speech improved considerably. Next day morning, his speech was clear, full of sentences, although the volume was less and it was non-spontaneous. There was a continuous improvement in his speech, and it was almost normal after the third day of the ECT session. Further, ECT sessions were withheld. The patient was kept in hospital for further 1 week. After satisfaction of the treating team, the patient was discharged with a PANNS score of 46 and a GAF score of 61-70. This patient is on continuous outpatient follow-up for past 3 months and is doing well on olanzapine 20 mg per day. This patient recently attended a program dedicated to the earning of livelihood by the recovered patients of chronic psychiatric illnesses.

## Discussion

Mutism is most commonly associated with catatonia. However, there are several other psychiatric disorders such as schizophrenia, mania, depression, and dissociative disorders that are known to cause mutism. There are also organic causes for mutism like dementia, neurodegenerative and demyelinating disorders, head injury, encephalitis, frontal lobe lesions, postictal phase of epilepsy, laryngeal tumors, and endocrine disorders, e.g. hyperparathyroidism.<sup>[7]</sup> Also, certain medications can lead to mutism for e.g. tacrolimus and cyclosporine.<sup>[8,9]</sup>

In the western literature, it has been discussed that mutism is rarely seen in schizophrenia and is mostly

associated with catatonia.<sup>[7]</sup> In our index case after the detailed workup, we did not find any organic cause for mutism, and there were no other catatonic signs or any significant negative symptoms that ruled out catatonia. So, the most likely explanation of mutism in our case could be due to positive psychotic symptoms, i.e. secondary to paranoid delusion belief. To our knowledge, there are only few studies that have reported non-catatonic mutism in schizophrenia and our case also highlights the same.

The other significant finding in our case was dramatic response of mutism to single ECT session. To best of our knowledge, this is the first case report showing such a dramatic response of non-catatonic mutism to just a single ECT session in combination to antipsychotic medications. Earlier, there have been two case reports that have mentioned about response of ECT. Basu *et al.* reported improvement of a non-catatonic mutism of 3 years in a schizophrenic patient to 14 sessions of ECT.<sup>[5]</sup> Grover *et al.* reported improvement of a similar patient on continuation ECT after being mute for 2 years.<sup>[6]</sup>

Despite the fact that ECT is safe and effective method in suitable patients, and is an important psychiatric treatment, particularly for severe or refractory cases, ECT is usually used as a second-line treatment. This is mainly because of social stigma and excessive concern about its side effects particularly potential retrograde amnesia.<sup>[10]</sup> However, seeing the dramatic response with single session of ECT in our case, we were compelled to think that ECT may be of particular value for patients having non-catatonic mutism not responding to other treatments.

All three cases of long-lasting mutism, including ours, did not respond to antipsychotics alone and were proved to be treatment-resistant cases. May be combining ECT with antipsychotics prove effective and efficacious in mitigating the paranoid delusion, and indirectly patient starts communicating verbally.<sup>[9]</sup> On this basis, we may hypothesize that the presence of non-catatonic mutism may be a positive indicator for response to ECT in such cases.

However, there remains one unanswered question, i.e. how is it that single ECT session could relieve non-catatonic mutism so rapidly? The mechanism is still unknown. Explaining the effect of single ECT is little difficult, but the role of concurrent anti-psychotic use cannot be ignored. The possible explanation of the synergistic effect seen with the augmentation of ECT on antipsychotic treatment is that the seizure induced by single session of ECT changes the blood-brain barrier (BBB) permeability; this leads to the passage of large

molecules across blood vessels into the CNS.<sup>[11,12]</sup> This allows a greater amount of antipsychotic drug to enter brain tissue without affecting tissue concentrations in other organs. As we know the effectiveness of antipsychotic drugs is dose dependent, thus the change in BBB permeability allows greater amounts of antipsychotic drug to enter brain. In this way, combination therapy with ECT and antipsychotic drug results in a synergistic effect. Is there a synergism in this case? Are there some predictors of this response?

Further, all such presentations in the recent past have been reported from Indian subcontinent only, none of them being from western world.<sup>[4-6]</sup> The presumptions for presentation could be a cultural/religious acceptance of such states as a coping strategy such as 'Manu Vart'. The long duration of untreated psychosis, possibly due to limited resources akin to pre-neuroleptic era of western world could explain such long mutisms in India as was prevalent in Micronesian islands.

Very few studies and case series have described non-catatonic mutism in schizophrenia. This case points towards the possible and early use of (augmentation of) ECT if a subject with schizophrenia presents with mutism and does not respond to antipsychotic medications and thereby improve prognosis. Further studies needs to be done to replicate this finding.

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