Catatonia Development in a Schizoaffective Patient following Electroconvulsive Therapy

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Catatonia is a syndrome that can be treated with electroconvulsive therapy (ECT). Hence, onset of catatonia during treatment of patients with ECT is not expected. In this manuscript, a schizoaffective disorder patient with a positive history of traumatic brain injury and no consumption of benzodiazepines is being reported, who became catatonic during ECT.

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Introduction

Criteria of Diagnostic and Statistical Manual of Mental Disorders, published by the American Association (DSM-IV), for Catatonia syndrome is as follows: Motor immobility which is evidenced by catalepsy (including waxy flexibility) or stupor, excessive motor activity (which is apparently purposeless and is not influenced by external stimuli), extreme negativism (which is an apparently motiveless resistance to all instructions or maintenance of a rigid posture against all attempts to be moved) or mutism, peculiarities of voluntary movements evidenced by posturing (voluntary assumption of inappropriate or bizarre postures), stereotyped movements, prominent mannerisms, or prominent grimacing and echolalia or echopraxia (1).

Catatonia can be treated with electroconvulsive therapy (ECT), benzodiazepines or barbiturates (2). ECT can also be a treatment for life-threatening depression or anti-depressant refractory major depressive disorder, catatonia, difficult-to- treat patients with acute mania, mixed mania, schizoaffective state or schizophrenia (3). Hence, onset of catatonia during the course of electroconvulsive therapy is unexpected and paradoxical (4). However, we are going to describe a schizoaffective patient with a positive history of head trauma and no consumption of benzodiazepines who became catatonic during ECT.

Case Report

A 23-year-old man was referred with aggression, odd behavior, thought control delusion and auditory hallucination. His symptoms commenced about 4 years ago. He made a suicide attempt and was hospitalized with head trauma (amnesia for 3 hours and loss of consciousness for half an hour; without necessity of ICU admission), vertebral column and foreleg fractures 3 years ago. He had received psychiatric medications during the last 2 years. His symptoms recurred sometimes and intensified during the last 6 months before the admission.

Besides, he had a history of one year consumption of haloperidol (7.5 mg daily) and lithium carbonate (900 mg daily). Furthermore, olanzapine and biperiden tablets were prescribed for him twice a day. Moreover, his

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psychiatric history was significant because of multiple admissions for schizoaffective disorder.

At this time of admission, his attitude was not cooperative, his mood was irritable with congruent affect, and his speech was high tone. He reported voices from the sky, while he was alert and well oriented. During his admission, he suffered from insomnia and disturbed other patients at nights. His medical examinations were unremarkable. Brain MRI was performed and it was accompanied with no space occupying lesion or other abnormalities.

He continued to take aforementioned drugs. Then, haloperidol and lithium carbonate doses were increased to 20 mg and 0.9 mg/dl of serum level respectively, within 3 weeks of admission. Because of unresponsiveness, lithium carbonate was changed to carbamazepine. Despite the implemented changes in drug regimen, this patients' mood did not alter after 3 weeks. Consequently, these drugs were withdrawn within a week, except haloperidol. Thereafter, a daily treatment with ECT started, which was administered bi-temporally with a THYMATRON^{TM} device and energy of 30% to 40%. Seizures lasted between 24 to 75 seconds. Anesthesia was induced immediately before ECT by atropine 0.5mg IV, succynilcholine 30mg IV and nesdonal 200mg IV.

After the 5th session of ECT his irritability intensified, and after the 6th session he stared vacantly with waxy flexibility, negativism and posturing. These symptoms were not accompanied by hyperthermia, extra pyramidal symptoms, altered mental status or autonomic dysfunctions which are cardinal symptoms of NMS (5). The other examinations were unremarkable. Based on the signs, diagnosis of catatonia was confirmed. ECT was discontinued, haloperidol was stopped and lorazepam 2 mg/day was prescribed for this patient, which was increased to 4 mg/day 2 days later. Furthermore, low doses of lorazepam were prescribed to control the irritability and prevent the probable delirium. Lorazepam dose was increased to 4 mg/day after evolution of fulminant catatonia. The signs of catatonia were resolved during 3 days. Accordingly, lorazepam was tapered during 7 days after catatonia remission. Then, clozapine (25 mg daily) was started, and its dose was escalated to 250 mg daily during one month till patient's discharge.

The patient was discharged 4 weeks later, when his symptoms were resolved. Moreover, he has had no delusion, hallucination or irritability during the recent 6 months.

Discussion

In this manuscript, a patient with approximately 4 years of recurrent symptomatic schizoaffective disorder, a positive history of head trauma, a normal MRI and a negative history of benzodiazepines consumption or cessation is reported, who became catatonic after the 6th session of ECT. Interestingly, catatonia symptoms were resolved after discontinuation of ECT and oral administration of lorazepam. ECT has been used as one of catatonia treatments for about 70 years and during this period several studies have confirmed its effectiveness (6-8). Hence, catatonia commencement during ECT is not expected. Nonetheless, there are reports on some cases that reveal this paradox. Giving patients ECT immediately after cessation of benzodiazepines (4) and discontinuation of mixed sedatives (9-11) were mentioned as probable causes of catatonia development in these cases.

In the present described case, catatonia appearance can be attributed to two suspected elements. The first probable etiology is this patient's positive head trauma history. Although no evidence of bleeding and macro-structural abnormalities were observed at the time of trauma, the 3 hours history of amnesia in addition to the 30 minutes loss of consciousness can make micro and cytoarchitectural changes probable, which may explain the onset of catatonia during ECT. Indeed, this statement needs more researches to be approved.

The second probable etiology is an increased concentration of haloperidol in Central nervous system (CNS). Blood brain barrier permeability increases following ECT (12). As high dose haloperidol was prescribed

for this patient, the abovementioned increased permeability might have resulted in an increased haloperidol concentration in CNS. This phenomenon may have caused induction of haloperidol side effects such as catatonia (13), although other side effects of Haloperidol were not observed.

In conclusion, considering the present case, ECT administration to patients with history of traumatic brain injuries should be taken care of more attentively.

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