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ORIGINAL PAPER

Response rate of catatonia to electroconvulsive therapy and its clinical correlates

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Abstract Electroconvulsive therapy (ECT) is an important treatment for catatonia. We aimed to study the response rate of catatonia treated with ECT and its clinical correlates in a large sample of inpatients. The ECT parameters of all patients ($n = 63$) admitted with catatonia between the months of January and December 2007 were examined. The number of ECTs administered, seizure threshold, failure to achieve adequate seizures and clinical signs pertaining to catatonia were analyzed. Response was considered as complete resolution of catatonic symptoms with Bush Francis Catatonia Rating Scale (BFCRS) score becoming zero. ECT was mostly started after failed lorazepam treatment except in 6 patients where ECT was the first choice. Patients who responded in 4 ECT sessions were considered fast responders (mean session number for response is 4 sessions) and response with 5 or more ECTs was considered slow response. Fast responders had significantly lower duration of catatonia (19.67 ± 21.66 days, $P = 0.02$) and higher BFCRS score at presentation (17.25 ± 6.21, $P = 0.03$). Presence of waxy flexibility and gegenhalten (22.60% vs. 0%, $P = 0.01$) predicted faster response, whereas presence of echophenomena (3.2% vs. 24.0%) predicted slow response. The response rate to catatonia appears to be associated with the severity and duration of catatonia, and the presence of certain catatonic signs.

Keywords Electroconvulsive therapy · Catatonia · Lorazepam · Echophenomena · Waxy flexibility

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Introduction

Catatonia is a heterogeneous neuropsychiatric disorder that varies in etiology, presentation, course and sequelae. It was first described by Kahlbaum in 1874 as tension insanity [1]. It was initially conceptualized to be a subtype of schizophrenia but was subsequently found to occur not only with other psychiatric conditions but also with medical conditions and drug-induced and toxic states. Catatonia can be characterized by concurrent motor, emotional, vegetative and behavioral symptoms [2]. Various authors have put forth evidences that warrant a separate classification for catatonia [3]. There are more than 40 motor signs of catatonia, but the presence of two prominent features for 24 h or longer is sufficient to identify the syndrome [3]. Electroconvulsive therapy (ECT) and benzodiazepines have been found to be effective in the treatment of catatonia [4]. The available literature mandates the treatment of underlying cause wherever identifiable and recommends the use of lorazepam as a first-line treatment [5]. The treatment response of catatonia to lorazepam has been recorded in the literature [2, 6–10]. Recent treatment guidelines list this as effective in both acute and chronic catatonia [11]. Benzodiazepines have been reported to be effective in 70% of the cases, with lorazepam demonstrating the highest frequency of use and a wide range of response rates up to 80% complete response rate [5, 8, 12, 13].

ECT is recommended for refractory and malignant catatonia [3–5]. The role of ECT in catatonia is understood mainly from case series and reports [6, 14–17]. However, data from randomized clinical trials on the effectiveness of ECT are not available due to obvious factors like limited samples and feasibility to conduct such a study in a severe psychiatric emergency. A recent retrospective study on 27 patients with catatonia reported the treatment response with

ECT [18]. This study demonstrated nearly 60% improvement rate with certain predictors of response like younger age, presence of autonomic dysregulation at baseline, especially higher body temperature, daily ECT during the first treatment week and longer duration of electroencephalogram seizure activity at last ECT session. An earlier study with 22 patients reported a higher response rate of 93%, and the authors concluded that ECT is effective in the resolution of both cardinal (primary) and secondary signs of catatonia. No difference was noticeable between catatonia due to affective illness and schizophrenia in this study [19].

In this study, we sought to examine the rate and speed of response of catatonia to ECT and its clinical predictors in a large sample of inpatients.

Methods

Setting

National Institute of Mental Health and Neurosciences, Bangalore, is a tertiary care psychiatric institute in South India with a bed-strength of 550. Annually, about 500 patients are prescribed ECTs, of which most are inpatients. All patients are evaluated by a multi-disciplinary mental health team under the supervision of academic faculty. ICD-10 criteria are used to diagnose the psychiatric disorders [20]. Consistent with the practice in the rest of the developing countries, the need to reduce the number of days of hospital stay with the hope of rapid response forms an important indication for starting ECTs [21].

The ECT team consists of psychiatrists, anesthetists, ECT nurses, dedicated staff and a state-of-the-art ECT suite. Each patient undergoes a pre-ECT evaluation consisting of detailed psychiatric and medical history, clinical examination with particular emphasis on neuropsychiatric aspects, pertinent laboratory investigations and, where necessary, ECG as well as brain imaging. Seizure threshold is determined during the first ECT session by the titration method [22]. During the course of ECT, if seizures are not elicited at electrical stimulus that was used during an earlier session, then the new threshold is determined by titration method again, starting from the previously used electrical dose. Treatment is administered using a NIV-IQURE machine (Technonivilak, Bangalore, India). Brief-pulse stimulus is delivered with constant current at 800 mA, with a frequency of 125 pulses/s (62.5 Hz) and pulse width of 1.5 ms; the duration of train is altered to adjust the dose. All ECTs are administered under anesthetic modification (thiopentone 3–4 mg/kg and succinylcholine 0.5–1 mg/kg). Cuff-method is used to record the duration of motor seizures. The details of indications for ECT, seizure threshold, duration of seizures and ECT-related complications are documented in the case-records. Changes in the clinical picture of the patients are recorded by the nurses, psychiatry postgraduate resident doctors, senior registrars and consultant psychiatrists. The referring psychiatrists decide on the number of ECTs for each patient—the reason for stopping ECT (clinical improvement/complication/withdrawal of consent, etc.) is noted in the file.

Sample

The ECT parameters of all patients admitted with catatonia between the months of January and December 2007 were examined retrospectively. The patients with catatonia are admitted routinely as a hospital practice in intensive observation ward. During this time, a complete history, physical and neurological examinations, mental status examination and the basic laboratory examinations are carried out. Then, all the cases are discussed with the senior residents. Finally, all the cases are again reviewed and discussed by a consultant psychiatrist on the teaching faculty of the institute. Bush Francis Catatonia Rating Scale (BFCRS) is used to assess the severity at the baseline and to measure improvement with treatment as a part of clinical protocol in the hospital. In cases where BFCRS is not applied, catatonic signs are examined regularly and recorded in the clinical sheets. Majority of the patients receive lorazepam as the initial treatment in the setting [10]. ECT is chosen as first-line treatment in morbidly ill patients with severe refusal of feeds to hasten up the recovery process or will be considered in patients who did not improve with lorazepam trial. All the patients received a uniform mode of ECT delivery in this sample: bitemporal electrode placement and weekly thrice ECT session frequency.

The number of ECT sessions received by the patients was used as a measure of speed of response, as the reason for stopping ECT was achievement of clinically significant improvement. An important reason for which ECT is prescribed in this setting is to shorten the hospital stay considering the morbidity associated with the illness. In this background, the number of days of hospital stay following initiation of ECT was also considered as an outcome measure. Other ECT-related parameters considered include total charge received, seizure threshold in sessions, number of failed attempts at seizure induction, number of failed ECT days and average motor seizure duration. Response was considered as complete resolution of catatonic symptoms and/or BFCRS score becoming zero. Improvement was considered as any clinical improvement and/or reduction of BFCRS scores during the treatment.

Statistical analysis was conducted using the Statistical Package for Social Sciences (SPSS) version 13.0 (SPSS

Inc., Chicago, IL, USA). Continuous variables were analyzed using the independent sample t test; categorical variables were analyzed using the Chi-square test. The level of significance was set at $P < 0.05$.

Results

Table 1 presents the clinical and ECT parameters of the whole sample ($n = 63$). The mean age of the sample was 26.12 years. The mean BFCRS score at baseline was 14.90. The mean duration of catatonia at presentation was 69.23 days. Since there was a significant skew in distribution of number of days of catatonia, median value of 10 days is depicted. About 59% of sample composed of women. The underlying psychiatric diagnosis is depicted in the Table 1. The mean number of ECT was 7.25 ± 2.54. ECT was given after a failed lorazepam trial in majority of the cases whereas only in 9.5% ECT formed the initial treatment of catatonia. Total days on lorazepam and number of days on lorazepam before ECT initiation are shown. The mean lorazepam dose received by the subjects was 5.83 ± 1.81. Response to ECT was noticed in 56 out of 63 patients (88.89%). Out of the rest 7 patients, improvement was not complete and they continued to have mild catatonic symptoms at discharge. There were 3 patients with affective disorder and 4 with non-affective psychosis among these. One of these patients received only 2 ECTs and took discharge before the ECT sessions were completed. The ECT parameters are shown in the same table.

Table 1 Clinical profile of patients who received ECT ($n = 63$)

	Mean ± SD or *N* (%)
Variable	
Age (years)	26.12 ± 9.35
Age of onset of illness	25.18 ± 8.99
Days of catatonia at presentation	111.85 ± 283.19
Duration of psychiatric illness (weeks)	61.64 ± 116.07
Gender—females	37 (58.7)
Catatonia as the first symptom of illness	23 (36.5)
Past history of catatonia	10 (15.9)
BFCRS score	14.90 ± 5.83
Diagnosis	
Schizophrenia	19 (30.2)
Major depression	16 (25.4)
BPAD	10 (15.9)
Psychosis NOS	8 (12.7)
Acute psychosis	6 (6.5)
Idiopathic catatonia (unspecified cause)	4 (6.3)
ECT as the first choice of treatment	6 (9.52)
Total number of days on lorazepam	20.22 ± 19.56
Number of days on lorazepam after which ECT given	5.98 ± 7.89
Number of ECT to show improvement	1.82 ± 0.92
Number of ECT to show response (present in 56/63 cases)	4.43 ± 2.03
Total charge received (milli coulombs)	804.07 ± 701.32
Mean number of ECTs per patient	7.25 ± 2.54
Mean charge delivered per ECT session	110.91 ± 21.14
Average motor seizure duration	60.45 ± 17.43
Thiopentone dosage (mg)	138.50 ± 30.72
Succinylcholine dosage (mg)	29.46 ± 6.78
Past history of receiving ECT	10 (17.5)
Use of anticonvulsant	1 (1.8)

Comparison of fast and slow responders

The mean number of sessions of ECT needed to produce the response (about 4, shown in Table 1) was taken as a cutoff to divide responders into fast and slow responders. Patients who responded in 4 ECT sessions were considered fast responders (mean session number for response is about 4 sessions) and response with 5 or more ECTs was considered slow response. Table 2 shows the clinical and ECT parameter differences between fast and slow responders. The slow responders had significantly greater number days of catatonia at presentation ($P = 0.02$). The fast responders appeared to have a significantly greater severity of catatonic symptoms as measured by BFCRS ($P = 0.03$), lesser electrical charge used overall, and shorter duration of inpatient stay.

A comparison of the baseline catatonic symptoms as scored in BFCRS and clinical records of patients between the groups was done and results are shown in Table 3. Waxy flexibility and gegenhalten significantly predicted faster response, while presence of echophenomena predicted a slower response.

Discussion

The evaluation of clinical and ECT parameters of 63 patients with catatonia in this sample reveals a response rate of nearly 90% which is in agreement to previous report that ECT is an effective treatment for catatonia [5, 15, 19, 23, 24]. However, in a recent study, complete improvement was noticed in only about 60% of the patients [18]. The mean age of the sample was 49 years in that study and the improvement was associated with younger age. In this study, the sample is much younger with a mean age of 26 years. The patients posted for ECT were lorazepam non-responders in most of the cases. Consistent with the

Table 2 Comparison between fast and slow responders—clinical and ECT parameters (total $n = 63$)

	Fast responders 31 (55.36%)	Slow responders 25 (44.64%)	*t*/Chi-square	*P*
Variable				
Mean age, years (SD)	27.22 (10.75)	25.52 (8.10)	−0.65	0.51
Days of catatonia at presentation	19.67 ± 21.66	130.68 ± 236.32	2.60	**0.02**
Gender—female	19 (61.3)	13 (52.0)	0.48	0.45
Past history of catatonia	5 (16.1)	5 (20.0)	0.14	0.70
Total days on lorazepam	27.82 ± 51.44	13.43 ± 13.13	−1.30	0.19
No of days on lorazepam before ECT	4.92 ± 3.77	7.17 ± 11.30	0.98	0.32
Mean BFCRS score	17.25 ± 6.21	13.05 ± 5.10	−2.25	0.030
Concurrent medications				
Lithium	4 (12.9)	0	3.47	0.06
Sodium valproate	0	1 (4)	1.26	0.26
Risperidone	10 (32.3)	10 (40)	0.36	0.54
Olanzapine	7 (22.6)	4 (16)	0.38	0.53
Chlorpromazine	1 (3.2)	0	0.82	0.36
Lorazepam	27 (87.2)	22 (88)	0.01	0.91
Diagnoses [*n* (%)]				
Affective illness	14 (46.67)	8 (34.80)	0.75	0.38
Psychotic illness	16 (53.30)	15 (65.21)		
Idiopathic catatonia	2 (6.44)	1 (4)		
Good response to past ECTs [*n* (%)]	6 (19.4)	4 (16.7)	0.72	0.69
Weight, kg (SD)	46.70 ± 11.79	45.16 ± 7.55	−0.56	0.57
Thiopentone mean dose, mg (SD)	136.45 ± 25.63	142.60 ± 36.00	0.76	0.45
Succinylcholine, mg (SD)	28.54 ± 6.08	30.62 ± 7.70	1.11	0.26
Concurrent use of anti-epileptics [*n* (%)]	0 (0)	1 (4)	1.26	0.44
Duration of inpatient stay after ECT initiation in days	18.38 ± 10.65	28.16 ± 11.91	3.23	0.002
Duration of inpatient stay before ECT initiation in days	4.87 ± 4.27	4.36 ± 2.62	−0.52	0.60
Total charge in milli coulombs	632.90 ± 471.00	1024 ± 883.27	2.12	0.03
Threshold at first ECT in milli coulombs	66.77 ± 42.22	70.80 ± 37.62	0.37	0.71
Second threshold in milli coulombs	80.61 ± 52.32	85.20 ± 44.73	0.34	0.73
Third threshold in milli coulombs	90.00 ± 51.38	86.40 ± 45.26	−0.27	0.78
Number of failed attempts to induce seizures	1.32 ± 1.04	1.44 ± 1.26	0.38	0.70
Average duration of motor seizure in seconds	63.79 ± 20.50	57.04 ± 11.99	−1.45	0.15

Bold value is statistically significant

$P < 0.05$ significant

literature, ECT is a good and effective option for these patients [4, 6, 17, 25]. ECT was considered as a first option even before lorazepam treatment in 6 cases where gravity of the condition such as extremely poor food intake and extremely poor general health prompted initiation of ECT for rapid relief of symptoms. The diagnosis of patients varied as shown in the Table 1. As it is shown in this study, the rapid alleviation of the epiphenomenon of catatonic symptoms does not depend on the underlying diagnosis. As shown in Table 2, seizure thresholds rise progressively with ECT in catatonia [26]. The response of catatonia to both benzodiazepines and ECT could suggest that catatonia can be the result of abnormal brain electrical/seizure activity. The mean number of ECTs is also lesser in our sample (mean = 7.25) compared to a recent study which needed a mean number of 14.6 ECTs for improvement [18].

The fast responders were the ones with a shorter duration of illness illustrating the fact that early detection of illness plays a crucial role in treatment response. This is similar to the findings by van Waarde et al. [18] where improved group had less number of days of catatonia at presentation. In the present study, intensity and severity of catatonia also played a role in response to treatment. Faster treatment response was associated with higher rating in BFCRS. Hence, an acute onset of catatonic syndrome with higher occurrence and severity of clinical signs predicted a faster response, whereas those patients with a chronic catatonic syndrome with milder symptoms responded

Table 3 Catatonic symptoms in relation to response rate

Catatonic symptom	Fast responder	Slow responder	T/chi value	*P* value
Excitement	4 (12.9)	2 (8.0)	0.88	0.64
Stupor	5 (16.1)	2 (8.0)	0.94	0.81
Mutism	30 (96.77)	23 (92.0)	0.89	0.82
Staring	25 (80.60)	20 (80.0)	1.32	0.98
Posturing	22 (70.96)	19 (76.0)	2.26	0.51
Grimacing	6 (19.35)	2 (8.0)	4.82	0.18
Stereotypy	2 (6.45)	2 (8.0)	2.07	0.55
Mannerism	3 (9.6)	2 (8.0)	4.90	0.08
Verbigeration	1 (3.2)	0	0.82	0.36
Rigidity	12 (38.70)	6 (24.0)	3.50	0.32
Negativism	21 (67.74)	19 (76.0)	1.40	0.70
Withdrawal of feeds	25 (80.46)	21 (84.0)	2.47	0.47
Waxy flexibility	7 (22.60)	0	6.45	**0.01**
Ambitendency	11 (35.5)	6 (24.0)	0.86	0.35
Echolalia/praxia	1 (3.2)	6 (24.0)	5.45 0.95	**0.01**
Impulsivity	1 (3.2)	1 (4.0)	2.04	0.35
Automatic obedience	4 (12.9)	3 (12.0)	1.88	0.59
Mitgehen	1 (3.2)	2 (8.0)	0.62	0.43
Gegenhalten	7 (22.6)	1 (4.0)	3.90	**0.04**
Grasp reflex	0	1 (4.0)	1.26	0.26
Autonomic abnormality	3 (9.6)	1 (4.0)	4.40	0.11

Bold values are statistically significant

$P < 0.05$ significant

slowly. This indicates the need to explore alternate strategies to treat this group of slow responders. Also, one needs to examine the neurobiological mechanism of relation between delay in treatment and ECT response rate.

A very interesting finding was a relationship between some of the catatonic signs and response rate to ECT in this study. Subjects with waxy flexibility and gegenhalten showed a faster response rate, while subjects with echophenomena showed a slower response. This intriguing observation on certain catatonic signs predicting response rate is difficult to explain at this juncture. However, it is essential for clinicians to be familiar with the reliably identifiable signs of the syndrome, given the fact that they could differentially predict the response rate. This also could be preliminary evidence that may help shed light on the understanding of motor regulatory systems and their changes during treatment. Catatonic patients who showed faster response stayed for a significantly shorter period in the hospital. There were no differences between the groups based on ECT parameters like average motor seizure duration, seizure thresholds and failed attempts at seizure induction. This again points at the important role of clinical variables, illness severity and illness duration as the major mediating factors of response rate.

The strengths of the study are that accurate recording of ECT parameters is present uniformly in all the patients. Additionally, as a protocol, majority of the patients had the standardized severity rating obtained at the baseline and treatment course. Certain limitations of this study need to be acknowledged. A major limitation is the retrospective review design. The baseline diagnosis was chart based. However, this limitation was partly addressed by an independent review of all the charts by two senior residents. Diagnosis was made for the patients in the file after detailed and meticulous evaluation by a junior resident and further confirmed by a senior resident and a consultant psychiatrist. Dosing limitation in the form of varying doses given for different subjects also needs to be acknowledged. This study adds to the literature with a large sample of catatonia patients that ECT forms a very important treatment for this psychiatric emergency and that certain clinical signs of catatonia could predict the response rate. Larger studies with prospective design would help in gaining an accurate estimation of the aforementioned factors of response rate. Similarly, imaging studies using magnetic resonance spectroscopy and SPECT to find the neurobiological substrates of ECT response in catatonia and its correlation with the baseline signs could be the future direction.

Conflict of interest None.

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