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Original Article

Does Substance Use Affect Electroconvulsive Therapy Outcome in Schizophrenia – Cohort and Descriptive Study

S Narmadha[1], S Malini[2], P Saravanakumar[3]

[1]Junior Resident, Department of Psychiatry, Institute of Mental Health, Chennai, Tamil Nadu, India, [2]Assistant Professor, Department of of Psychiatry, Institute of Mental Health, Chennai, Tamil Nadu, India, [3]Associate Professor, Department of Community Medicine, Kilpauk Medical College, Chennai, Tamil Nadu, India

Abstract

Introduction: Electroconvulsive therapy (ECT) has been used to treat schizophrenia, a chronic mental disorder characterized by symptoms such as delusions, hallucinations, disordered thinking, and abnormal behavior. This study aims to investigate the impact of substance abuse on ECT in patients with schizophrenia.

Methods: This prospective and cohort study was conducted at the Institute of Mental Health in Chennai over 1 year, with ethical committee approval. The study collected sociodemographic details such as age, gender, and others. The history of substance use was also documented, including information on the type of substances used. The baseline condition of the psychiatric illness of each patient was assessed using the brief psychiatry rating scale (BPRS) to determine the severity of symptoms.

Results: The study participants had an average age of 34.83 years with a standard deviation of 11.46. About 57.5% of them had a history of new substance use. The study's results showed a statistically significant difference in BPRS scores among patients who used cannabis, alcohol, nicotine, and tobacco before and after ECT. The BPRS scores for patients who used cannabis decreased from 52.71 ± 6.77 to 37.14 ± 10.27, and for alcohol, decreased from 50.78 ± 10.61 to 37.39 ± 11.19. For nicotine decreased from 51.00 ± 10.06 to 36.44 ± 10.73 and for tobacco decreased from 48.00 ± 11.28 to 37.58 ± 11.07.

Conclusion: It is important to note that the frequency and length of substance use determine the relationship between substance abuse and ECT treatment outcomes for schizophrenia patients.

Key words: Brief psychiatry rating scale, Electroconvulsive therapy, Schizophrenia, Substance use

INTRODUCTION

Psychiatric disorders like schizophrenia, affecting people of all backgrounds and socioeconomic statuses, are considered among the most debilitating worldwide.[1,2] In the world, 1% of the population suffers from schizophrenia. Electroconvulsive therapy (ECT), also known as ECT, was introduced in 1938 and used in psychiatry for over 75 years. After the introduction of psychotropic medications in the 1950s, their use declined but has increased in recent years. Due to the limited success of pharmacological treatments in some patients with depression and schizophrenia.[3-5] ECT is considered acceptable for major depression and is effective and safe, based on well-established research. According to some studies, up to 30% of patients with schizophrenia do not respond adequately to antipsychotic medication and experience persistent symptoms.[6,7]

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The Epidemiological Catchment Area study found that a high proportion of patients with schizophrenia have issues with substance abuse compared to the general population. Specifically, 47% of patients with schizophrenia have reported severe problems with drug or alcohol use at some point in their lifetime, while only 16% of the general population has reported similar issues. According to the Epidemiological Catchment Area study,

Corresponding Author: S Malini, Department of of Psychiatry, Institute of Mental Health, Chennai, Tamil Nadu, India.

47% of patients with schizophrenia have severe problems with drug or alcohol use during their lifetime, compared to only 16% of the general population.[8,9] According to several studies, between 60% and 90% of patients with schizophrenia are said to have smoked cigarettes at some point in their lives. The estimated lifetime prevalence range for alcohol use disorders is 21–86%. According to reports, the lifetime prevalence of cannabis use ranges from 17% to 83%, and that of cocaine use is between 15% and 50%.[10-15]

Genetic risk factors for schizophrenia, particularly those related to neural systems that contribute to psychosis and addiction, can make patients more susceptible to substance abuse. The "primary addiction hypothesis" suggests that the susceptibility to schizophrenia primes the reward circuits in the brain, increasing the risk of drug addiction after exposure to drugs. The "two-hit hypothesis" suggests that drug abuse combined with other genetic and environmental risk factors can contribute to the development of schizophrenia. These hypotheses highlight the complex relationship between substance abuse and schizophrenia.[16] This study aims to investigate substance abuse's impact on ECT in patients with schizophrenia. Substance abuse can complicate the treatment of schizophrenia and may affect the effectiveness of ECT. Examining the effect of substance abuse on ECT parameters may provide valuable information for improving the treatment of schizophrenia and addressing substance abuse.

METHODS

This prospective and cohort study was conducted at the Institute of Mental Health in Chennai over 1 year, with ethical committee approval. The study included 40 male patients between 18 and 60 diagnosed with schizophrenia based on the ICD-10 criteria and was indicated for ECT treatment. Patients were included in the study through consecutive sampling and were willing to consent.

Patients with associated intellectual disabilities and those with psychiatric illnesses other than schizophrenia were excluded from the study.

The aim of the study was to compare the brief psychiatry rating scale (BPRS) scores of patients before and after ECT treatment, with a calculated standard deviation of 1.17, 80% power, and 95% confidence level.

The study collected sociodemographic details such as age, gender, and others. The history of substance use was also documented, including information on the type of substances used. The baseline condition of the psychiatric illness of each patient was assessed using the BPRS to determine the severity of symptoms. This information is important in understanding the relationship between substance abuse and the effectiveness of ECT in treating schizophrenia.

Table 1: Distribution of substance use among the study participants (*n*=40)

Distribution of each substance use	Number of patients (%)
Alcohol	18 (45)
Cannabis	7 (17.50)
Nicotine	16 (40)
Tobacco	12 (30)

Descriptive statistics were reported as mean (SD) for continuous variables and frequencies (percentage) for categorical variables. Chi-square at a 5% level of significance was used to find statistical significance. Fischer's exact test is when the expected cell count is <5. Repeated measure ANOVA was used to find the significant difference between time points. Data were statistically evaluated with IBM SPSS Statistics for Windows, Version 20.0.

RESULTS

The total mean age of the study participants was 34.83 ± 11.46 years. Around 57.5% had new substance use. Among the 40 patients, 18 (45%) had a history of alcohol use, and 7 (17.5%) patients had a history of cannabis use. Sixteen (40%) patients had a history of nicotine use, and 12 (30%) had a history of tobacco use [Table 1].

Among the study subjects, there were about 45% (n = 18) between 19 and 30 years, 17.5% (n = 7) between 31 and 40 years, 30% (n = 12) between 41 and 50 years, and 7.5% (n = 3) between 51 and 60 years.

Among alcohol users, the majority were in 19–30 years (55.6%), followed by 16.7% in both 31–40 years and 41–50 years, and 11.1% in 51–60 years.

Among cannabis users, the majority were in the age group of 19–30 years (71.4%) followed by 14.3% in 31–40 years and 51–60 years.

Among nicotine users, majority were in 19–30 years (37.5%) followed by 31.5% in 41–50 years, 18.8% in 31–40 years and 12.5% in 51–60 years.

Among tobacco users, majority were in 19–30 years (58.3%), followed by 25% in 41–50 years, 8.3% in 31–40 years and 51–60 years, respectively [Table 2].

A repeated measures ANOVA with a Greenhouse-Geisser correction determined that mean BPRS differed statistically significantly among alcohol users between time points (F [1.818, 30.912] = 76.111, $P < 0.001$). *Post hoc* analysis with a Bonferroni adjustment revealed that BPRS was statistically significantly decreased from pre-ECT to post-ECT (13.389 [95% CI, 9.69–17.08], $P \leq 0.001$), and from pre-ECT to on termination (26.056 (95% CI, 21.17–30.94), $P \leq 0.001$).

A repeated measures ANOVA with a Greenhouse-Geisser correction determined that the mean BPRS score differed significantly among cannabis users between time points (F [1.867, 11.199] = 43.425, $P < 0.001$), meaning that the mean BPRS score changed over time and the changes were statistically significant.

Post hoc analysis with a Bonferroni adjustment was conducted to investigate the differences between time points further. The study revealed that the mean BPRS score was statistically significantly decreased from pre-ECT to post-ECT (15.571 [95% CI, 7.44–23.70], $P \leq 0.003$), and from pre-ECT to on termination (27.71 [95% CI, 21.26–34.16], $P \leq 0.001$).

BPRS differed statistically significantly among nicotine users between time points (F [1.648, 24.473] = 80.54, $P < 0.001$). *Post hoc* analysis with a Bonferroni adjustment revealed that BPRS was statistically significantly decreased from pre-ECT to post-ECT (14.56 [95% CI, 11.24–17.88], $P \leq 0.003$), and from pre-ECT to on termination (26.94 [95% CI, 21.86–32.01], $P \leq 0.001$).

BPRS differed statistically significantly among tobacco users between time points (F [1.653, 18.18] = 47.69, $P < 0.001$). *Post hoc* analysis with a Bonferroni adjustment revealed that BPRS was statistically significantly decreased from pre-ECT to post-ECT (10.41 [95% CI, 6.43–14.40], $P \leq 0.001$), and from pre-ECT to on termination (23.58 [95% CI, 17.43–29.73], $P \leq 0.001$) [Table 3].

Table 2: Distribution of age among substance abuse

Age	Alcohol (%)	Cannabis (%)	Nicotine (%)	Tobacco (%)
19–30	10 (55.6)	5 (71.4)	6 (37.5)	7 (58.3)
31–40	3 (16.7)	1 (14.3)	3 (18.8)	1 (8.3)
41–50	3 (16.7)	0	5 (31.5)	3 (25)
51–60	2 (11.1)	1 (14.3)	2 (12.5)	1 (8.3)

Table 3: Distribution of BPRS pre- and post-electroconvulsive therapy among the substance use

Substance use	Mean±SD	95% CI	*P*-value
Alcohol			
Pre-ECT	50.78±10.619	45.497–56.059	-
Post-ETC	37.39±11.194	31.822–42.956	<0.001
On termination	24.72±5.278	22.097–27.347	<0.001
Cannabis			
Pre-ECT	52.71±6.77	46.448–58.980	-
Post-ETC	37.14±10.27	27.645–46.641	0.003
On termination	25.00±4.16	21.150–28.850	<0.001
Nicotine			
Pre-ECT	51.00±10.06	45.636–56.364	-
Post-ETC	36.44±10.73	30.718–42.157	<0.001
On termination	24.06±5.18	21.301–26.824	<0.001
Tobacco			
Pre-ECT	48.00±11.282	40.832–55.168	-
Post-ETC	37.58±11.07	30.547–44.619	<0.001
On termination	24.42±6.03	20.581–28.252	<0.001

DISCUSSION

Mental illnesses like schizophrenia and substance abuse are common and debilitating conditions that frequently coexist in the same person. Suicidal ideation and behavior are significantly increased in people with schizophrenia who abuse alcohol and other substances. The impact of alcohol on the brain is complex and influenced by multiple variables such as the amount consumed, age of first use, duration of use, personal characteristics, and health conditions.[16] In individuals with alcohol dependence, there is likely to be more significant brain damage than in those with alcohol abuse.

Research has shown that chronic alcoholics have a greater amount of brain shrinkage than healthy individuals, especially in brain regions such as the prefrontal cortex and limbic system, as seen in imaging studies and after-death examinations. Alcohol consumption can also lead to changes in neurotransmitters, particularly glutamate. Chronic alcohol use has been shown to increase the number of glutamate receptor sites in the hippocampus, which can become overstimulated and contribute to brain damage.[17]

In our findings, we have observed that BPRS was statistically significantly decreased from pre-ECT to post-ECT (13.389 [95% CI, 9.69–17.08], $P \leq 0.001$), and from pre-ECT to on termination (26.056 [95% CI, 21.17–30.94], $P \leq 0.001$). Our finding was consistent with the result of Moss *et al.*, with no difference in ECT outcome between those with comorbid alcohol abuse and those without based on a percent decrease in pre- and post-ECT symptom scores (abuse: mean [SD], 0.89 [0.2] vs. non-abuse: mean [SD], 0.93 [0.16]; Wilcoxon, 1332; $P = 0.086$).[18] Cannabis use is prevalent among individuals

with schizophrenia and can have adverse effects on the course of the illness, including increased symptoms and decreased cognitive function.[19]

Smoking cigarettes has decreased serotonin levels in the brain and inhibiting monoamine oxidase. It leads to increased impulsivity and suicidality in individuals with mental health conditions such as schizoaffective disorder.[20] In the present study, we also found a significant decrease in BPRS scores in cannabis, nicotine, and tobacco users from pre-ECT to post-ECT. The accordance of a study conducted by Nicole *et al.* showed that adolescents and young adults who found positive for substance use had greater improvement in depression/functioning (-0.37 ± 0.14, $P = 0.009$), interpersonal relationships (-0.27 ± 0.13, $P = 0.045$), and emotional ability (-0.27 ± 0.14, $P = 0.044$) domains after the fifth ECT treatment.[21]

It is critical to have therapy that is both effective and safe. Including more detailed alcohol and drug histories, brain imaging, cognitive testing, and family histories of alcohol and drug abuse, as well as structured pre-treatment and post-therapy symptom assessments, could be included in the future studies. In summary, this study discovered that patients with schizophrenia who also had comorbid alcohol or substance abuse showed improvement after receiving ECT. The presence of alcohol dependence, nicotine dependence, tobacco usage, and cannabis dependence all impact the outcome; furthermore, this finding was found to be statistically significant in our study.

CONCLUSION

ECT is a highly effective treatment option for schizophrenia and substance abuse. The frequency with which patients use substances and the length of time that they have been sick before receiving ECT is important factors in determining the relationship between substance use and how well psychotic patients respond to ECT treatment. Further research is recommended to determine whether the duration of the current illness and the amount of substance use impact the effectiveness of ECT therapy in schizophrenia patients.

Limitations

The distinction between alcohol abuse and dependence is made through clinical assessment and examination, lacking objective measures of alcohol, and substance consumption at the time of the study. The impact of alcohol on the brain is diverse and subject to numerous variables such as the amount consumed, when it was first consumed, how long it was consumed, the individual's age, gender, family background, exposure to alcohol before birth, and any accompanying health conditions. Although complete information was not available in the study population, it is believed that individuals with alcohol dependence may have greater brain damage and dysfunction compared to those with alcohol abuse.

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