Etomidate versus Propofol for Motor Seizure Duration during Modified Electroconvulsive Therapy

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Abstract

Background: Certain anesthetic agents on account of their anticonvulsant property have a negative impact on motor seizure duration. Etomidate and propofol being devoid of the strong anticonvulsant property may be beneficial for use in electroconvulsive therapy (ECT). ECT requires sedation with a short-term anesthetic agent that does not interfere with seizure activity and has rapid onset and recovery to facilitate fast-tracking. Aims: The primary objective of this study was to compare motor seizure duration, and the secondary objective was to compare induction time, hemodynamic parameters, recovery time, and adverse effects between propofol and etomidate in modified ECT. Settings and Design: This is a prospective, double-blind, randomized, controlled study conducted in the Department of Anesthesia and Intensive care in a tertiary care hospital during 2018-2019. Materials and Methods: After ethical clearance from institutional ethics committee and written informed consent, a total of 70 patients, aged 18–65 years were randomly allocated using computer generated random number list into two groups - Group A - Propofol (1%) - 1.0 mg.kg⁻¹ and Group B - Etomidate 0.2 mg.kg⁻¹ as an intravenous induction agent. Intraoperatively, motor seizure duration, induction time, and hemodynamic parameters and at the end of procedure recovery parameters were assessed. Statistical Analysis Used: Data were described in terms of number (%) and mean ± standard deviation. Comparison of quantitative variables between the study groups was done using Student t-test and Mann Whitney U test for parametric and nonparametric variables respectively. For comparing categorical data, Chi-square (χ²) test was performed. Results: Mean motor seizure duration with etomidate (55.17 ± 19.06 s) was longer as compared to propofol (27.80 ± 17.33 s), and the difference was highly significant (P < 0.001). Among hemodynamic parameters, there was a significant increase in heart rate (P = 0.016) and significant fall in mean arterial pressure (P = 0.005) after induction with propofol as compared to etomidate. Conclusion: Etomidate has the advantage of longer seizure duration and stable hemodynamics. It can be a useful alternative in patients achieving suboptimal therapeutic responses to ECT or where seizure duration is too short.

Keywords: Electroconvulsive therapy, etomidate, motor seizure, propofol

INTRODUCTION

Electroconvulsive therapy (ECT) is a beneficial curative technique in the clinical practice of psychiatry.[1] Its indications include bipolar mood disorders, schizophrenia, catatonic psychosis, and delirium. Numerous studies in the literature have revealed a positive association during duration of seizure and therapeutic efficacy.[2,3] Patients experiencing motor seizure duration of <15 s achieve a less favorable response to ECT while a longer seizure duration does not provide any conspicuous therapeutic benefit.[4] The effectiveness of ECT in the amelioration of symptoms of various psychiatric ailments like catatonia, depression and schizophrenia relies on elicitation of generalized seizures which in turn leads to increase in brain derived neurotrophic factor (BDNF). The latter is a protein that assures survival and growth of new synapses and neurons thus, inducing neurogenesis and synaptogenesis in the hippocampus. Impaired neurogenesis may lead to severe depression. ECT is a useful remedy for normalization of neuroendocrine dysfunction in melancholic depression. Subconvulsive seizures may produce cognitive impairment...
without any therapeutic benefit.\[^{[3]}\] Since the measurement of seizure duration is one of the useful markers for therapeutic effectiveness. Hence, this study was undertaken to compare propofol and etomidate in modified ECT as regards motor seizure duration.

**Materials and Methods**

This was a prospective, randomized, double-blind, controlled study undertaken over 16 months from July 2018 to October 2019 after obtaining prior approval from Institutional ethics committee (IEC) in a tertiary care center with approval number REF/2018/06/02046, CTRI/2018/08/015218. Written informed consent was obtained from all patients included in the study via IEC approved consent form. A total of 70 patients of either gender in the age group of 18–55 years of the American Society of Anesthesiologists (ASA) physical status Classes I and II admitted to the psychiatry department scheduled to undergo ECT under general anesthesia were enrolled in this study. Exclusion criteria included patients with known allergy to the study drug, neuromuscular disorders, hypertension and other cardiovascular disorders, epilepsy and hypopituitarism. The patients were randomized by using a computer generated random number list and randomly allocated in the ratio of 1:1 to one of the two study groups using coded and sealed opaque envelopes that were opened shortly before induction of anesthesia. Two first authors enrolled the participants and were not involved in data collection. An independent observer, blinded to the type of drug being used, recorded the data. Group A patients received intravenous (i.v.) propofol (1%) - 1.0 mg.kg\(^{-1}\) (10 mg.mL\(^{-1}\) i.v. preparation, Neon Laboratories Limited, India). Group B patients received i.v. etomidate - 0.2 mg.kg\(^{-1}\) (2 mg.mL\(^{-1}\) i.v. preparation, Celon Laboratories Private Limited, India).

To avoid bias, both the drugs were prepared by an unknown investigator. All the patients were kept fasting overnight and continued antipsychotic treatment until the day of the procedure. After taking the patient in the operative room, IV line was secured, and standard intraoperative monitors such as electrocardiogram (ECG), noninvasive blood pressure, and pulse oximeter were attached for monitoring heart rate (HR), mean arterial pressure (MAP), and oxygen saturation (SpO\(_2\)). All the patients were premedicated with injection glycopyrrolate 0.2 mg i.v., injection ondansetron 4 mg i.v., and Injection tramadol 25 mg i.v. Patients were pre oxygenated with 100% oxygen for 3 min and after that general anesthesia was induced with i.v. anesthetic agent as per the group allocated till loss of verbal contact. Following this, electronic tourniquet cuff was applied to the lower leg and inflated to isolate the circulation of the foot to permit an accurate assessment of the motor seizure. Succinylcholine 0.5 mg.kg\(^{-1}\) i.v. was administered to all the patients for neuromuscular relaxation. When fasciculations subsided, bite block was inserted to prevent tongue bite. The psychiatrist was allowed to place bitemporal ECT electrodes on the forehead. A brief pulse stimulus of 1.0 s using a pulse of 70 Hz and 120 mC by BPE-2000 A machine was given to produce seizures. Subsequently, all the patients were ventilated with a face mask with 100% oxygen until the return of spontaneous respiration. All the patients were monitored for changes in HR, MAP, arterial SpO\(_2\), ECG changes, and respiratory rate before induction of anesthesia, after the administration of study drug, after giving succinylcholine, after applying ECT, at 1, 3, 5, 10 min, and then every 5 min throughout the procedure till shifting of the patient to post-anesthesia care unit (PACU).

Induction time was recorded as the time from injecting i.v. anesthetic agent till loss of verbal contact. The duration of motor seizure was recorded as the time from the ECT stimulus to the cessation of tonic–clonic motor activity in the isolated foot using a handheld stopwatch. Recovery parameters - (i) time to return of spontaneous ventilation (in minutes) was assessed as the time from i.v. administration of anesthetic agent until the appearance of the patient’s breathing efforts in the bag attached to the anesthesia circuit. (ii) Duration of recovery (in minutes) was assessed as the time from i.v. administration of anesthetic agent to the time taken to obey commands such as eye-opening. The postoperative assessment was done in PACU with emergence agitation score graded as (i) sleeping, (ii) awake and calm, (iii) irritable and crying, (iv) inconsolable crying, and (v) severe restlessness and disorientation; and patient satisfaction score graded as (i) pleased and calm patient, (ii) patient without any complaints (satisfaction is not bad), (iii) patient has some complaints, (iv) patient claimed that treatment was unpleasant and did not want to undergo same technique anymore.\[^{[6,7]}\] Any side effects such as pain on injection site, fall in SpO\(_2\), below 90%, hypotension, i.e., fall in the MAP below 60 mmHg, nausea, vomiting, and myoclonus were noted.

**Statistical analysis**

Data were described in terms of range, mean ± standard deviation, frequencies (number of cases), and relative frequencies (percentages) as appropriate. Comparison of quantitative variables between the study groups was done using Student’s t-test and Mann–Whitney U-test for parametric and nonparametric variables, respectively. For comparing categorical data, Chi-square test was performed, and the exact test was used when the expected frequency was <5. A probability value (P value) <0.05 was considered statistically significant and P < 0.001 was considered highly statistically significant. All statistical calculations were done using Statistical Package for the Social Science version 21 statistical program for Microsoft Windows (IBM Corp., Armonk, N.Y., USA).

A post hoc power analysis was conducted using the software package, G*Power (Faul and Erdfelder 1992). The alpha level used for this analysis was 0.05, and beta was 0.20. The sample size was estimated from the results of the previous study using the seizure duration as the parameter, which is the primary outcome of our study.\[^{[8]}\] Our sample size came out to be 35 subjects per group at the power of 1 and with an effect size of 3.82 with 10% chance of error with α = 0.05, β = 0.20, and confidence interval of 95%.
RESULTS

Seventy patients were studied in two groups [Figure 1]. These patients were comparable concerning age, weight, gender, and ASA status [Table 1]. Among hemodynamic parameters, there was a significant increase in HR and a significant fall in MAP after induction with propofol as compared to etomidate ($P = 0.016$, $P = 0.005$) respectively [Figures 2 and 3]. However, no significant differences were observed in HR and MAP in the rest of the procedure. Induction time was relatively more with etomidate as compared to propofol, but the difference was not statistically significant ($P = 0.234$) [Table 2]. In the propofol group, mean motor seizure duration was 27.80 (17.33) seconds and it was 55.17 (19.06) seconds in etomidate group and the difference was highly significant ($P < 0.001$) [Figure 4]. The return of spontaneous ventilation and recovery time was faster with propofol as compared to etomidate but the difference was not statistically significant [Table 2]. In the propofol group, the incidence of nausea and vomiting was 2.9% as compared to 5.7% in the etomidate group ($P = 0.555$). In the propofol group, the incidence of tachycardia was 31.45% as compared to 34.3% in the etomidate group ($P = 0.799$). Myoclonus was observed only in the etomidate group in 14.3% of patients ($P = 0.054$). No incidence of hypotension, bradycardia, hypoxemia, or pain on injection was observed in any group.

DISCUSSION

ECT represents a very challenging task for an anesthesiologist. The goal of modified ECT is to induce a generalized seizure which contradicts the anticonvulsant concept of general anesthesia. Almost all anesthetic agents have anticonvulsant properties because of their effects on the gamma-aminobutyric acid receptors and may, therefore, influence seizure variables and clinical outcome of ECT. The current study aimed to compare two induction agents, i.e., propofol and etomidate as they are currently being used for ECT in terms of their effect on motor seizure duration. In the present study, motor seizure duration was longer with etomidate as compared to propofol and the difference was statistically significant ($P < 0.001$) [Table 2 and Figure 4]. The elevation of seizure threshold after propofol administration may explain the lower duration of the seizure.\(^8\) Etomidate reduces the seizure threshold and is associated with longer seizure duration and may be helpful in a patient with short seizure time (<20 s) despite a maximal electrical stimulus.\(^9\) Our results are consistent with a study conducted by Mir et al. and Canbek et al. in which mean motor seizure duration was significantly longer in etomidate as compared to propofol group ($P < 0.05$).\(^10,11\) Our results are also similar to a study done by Gazdag et al. in which propofol (1 mg.kg\(^{-1}\)) and etomidate (0.2 mg.kg\(^{-1}\)) were compared during the ECT of patients with schizophrenia based on their impact on seizure activity and seizure-induced hemodynamic reactions. Both motor (EMG; $P = 0.006$) and electroencephalographic ($P = 0.014$) seizure duration

![Figure 1: Consort flow diagram](image1)

![Figure 2: Comparison of heart rate at different time intervals](image2)

### Table 1: Patient characteristics

|                   | Group A          | Group B          | $P$  |
|-------------------|------------------|------------------|------|
| Age (years)       | 31.34±9.83       | 30.77±8.25       | 0.793|
| Weight (kg)       | 61.57±12.50      | 63.71±12.84      | 0.482|
| ASA grade         |                  |                  |      |
| I                 | 27               | 25               | 0.584|
| II                | 8                | 10               |      |
| Gender            |                  |                  |      |
| Male              | 12               | 13               | 0.803|
| Female            | 23               | 22               |      |

All variables except ASA status and gender are expressed as mean±SD.

Group A stands for the group that received propofol, Group B stands for the group that received etomidate. ASA=American Society of Anesthesiologists, SD=Standard deviation.
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was significantly shorter after propofol than etomidate.[12] Avramov et al., in their study on the duration of seizure activity and cognitive recovery profiles after different doses of methohexital, propofol, and etomidate found that the duration of motor seizure was longest after etomidate and shortest after propofol ($P < 0.05$).[13] Etomidate is the only induction agent among all currently available induction agents (except ketamine) that may reduce the seizure threshold.[14] It has been suggested that propofol may be useful when patients, especially adolescents and young adults, have a very low seizure threshold or prolonged seizures. Alternatively, etomidate may be indicated when seizures are too short and possibly subtherapeutic despite maximum stimuli.[15] The quality of seizure is a function of the duration of seizure and the extent of central inhibition. Etomidate is more favorable than propofol concerning central inhibition. In light of its lack of anticonvulsant property, etomidate might seem preferable to propofol in ECT anesthesia in terms of seizure quality.[16] The induction was rapid with propofol as compared to etomidate but the difference in induction time was statistically insignificant ($P = 0.234$) [Table 2]. These results were comparable to the study conducted by Mir et al. who also found shorter induction time with propofol as compared to etomidate.[10] This might be because propofol has a rapid onset of action and rapid metabolism in the liver. The elimination half-life of propofol is 1.8 H and it has a very high clearance rate which accounts for its shorter induction time.[17,18] In the present study, comparing propofol and etomidate, induction was smoother with propofol and it was observed that propofol reduced the hypertensive response to ECT. However, it also caused tachycardia at induction. Preoperatively, both the groups were comparable as regards mean HR and MAP. After induction, there was a significant fall in MAP and a significant increase in HR in the propofol group as compared to the etomidate group ($P = 0.016$). The hypotension caused by propofol can be attributed to the reduction of the sympathetic activity causing vasodilatation or its direct effect on vascular smooth muscles.[19] The mechanism of the cardiovascular disturbances is the result of intense stimulation of the autonomic nervous system and a large increase in circulating catecholamines.[10] In the current study, MAP was reduced at an induction in the propofol group whereas in the etomidate group, it was comparatively stable. While propofol induces both unconsciousness and amnesia, it has no analgesic properties. Hence, the patient may exhibit a physiological response to the stress of painful stimuli. This possibly explains the tachycardia after induction.[20] Etomidate at induction doses typically produces a small increase in HR and little or no decrease in blood pressure. It has little effect on coronary perfusion pressure and reduces myocardial oxygen consumption. Thus, of all induction agents, etomidate is best suited to maintain cardiovascular stability especially in patients with coronary artery disease, cardiomyopathy, valvular heart disease, cerebral vascular disease, or hypovolemia.[21] Hemodynamic stability observed with etomidate may be due to its unique lack of effect on the sympathetic nervous system and baroreceptor functions.[22] In various studies, etomidate has shown less cardiovascular depression and it minimizes the use of vasopressor agents as compared to other induction agents in sepsis and critically ill patients.[23,24] Zahavi and Dannon in his study also found stable hemodynamics with etomidate as opposed to an elevation in blood pressure with other treatment groups (propofol and thiopental). In the present study also, hemodynamics were stable with etomidate.[23] Similar results were contemplated in studies conducted by Aggarwal et al., and Kumar on patients undergoing ECT comparing propofol and etomidate which showed a significant decrease in MAP and a significant increase in HR from baseline to induction in the propofol group as compared to the etomidate group.[22,26] These

![Figure 3: Comparison of mean arterial pressure at different time intervals](image)

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**Table 2: Comparison of induction time, motor seizure duration, and recovery time between two groups**

| Parameters                  | Group A     | Group B     | $P$   |
|-----------------------------|-------------|-------------|-------|
| Induction time (s)          | 39.80±3.13  | 50.00±2.74  | 0.000*|
| Motor seizure duration (s)  | 27.80±17.33 | 55.17±19.06 | 0.000*|
| Recovery time (min)         | 6.40±3.90   | 7.17±2.38   | 0.322 |

* $P<0.05$ significant. All variables are expressed as mean±SD. Group A stands for the group that received propofol, Group B stands for the group that received etomidate. SD=Standard deviation

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![Figure 4: Comparison of motor seizure duration in two study groups. The bottom and top of the box are first and third quartiles, the band inside the box is the median value, the whiskers represent the 10th and the 90th percentile and the black dots represent the outliers](image)
results were comparable to the present study. The propofol group had early and smooth recovery as compared to etomidate but the difference was statistically insignificant ($P = 0.322$). Our results are consistent with studies conducted by Shah et al., and Canbek et al., in which mean recovery time in terms of ability to obey vocal commands and eye-opening was shorter with propofol group as compared to thiopentone and midazolam groups ($P > 0.05$). Furthermore, the meantime to spontaneous respiration was shorter with propofol as compared to etomidate.$[8,10]$ The propofol has an onset time of 40 s and also the metabolism of propofol is rapid which occurs mainly in the liver accounting for its rapid induction and recovery.$[18]$ Avramov et al. in his study, in 1995, postulated that the duration of ECT induced seizure is the primary determinant of early recovery rather than the dose of the hypnotic drug. Hence, it explains the comparative faster recovery observed with propofol as compared to etomidate in the present study.$[13]$ Myoclonus defined as sudden jerks typically lasting for 10–50 ms, was observed with etomidate group and not with propofol group ($P = 0.054$). The possible cause of myoclonus is thought to be subcortical disinhibition. Interruption of the inhibitory synapses on the thalamocortical pathway also explains the myoclonic activity.$[17]$ Our results are consistent with the study conducted by Bisht et al. and Isitemiz et al. incidence of myoclonus was found to be reduced with opioid premedication which is consistent with our study results.$[28,29]$ The concerns regarding etomidate induced adrenal suppression have always been reported in the literature. However, a single dose of etomidate is unlikely to be associated with clinically relevant adrenal suppression.$[30]$ Patient satisfaction score and emergence agitation score were comparable in both groups and the difference was statistically insignificant ($P > 0.05$). No other adverse effect associated with any group was found in the present study.

The medications used for the treatment of the psychiatric patients cause sedation and may reduce the seizure threshold, but continuing treatment in these patients was indispensable for ethical reasons. Therefore, the interaction of psychoactive medications with propofol and etomidate could not be ruled out which constitutes a limitation of our study. We have not taken into account the possibility of dropouts or lost cases that constitutes one of our limitation. Seizure duration can be measured by several techniques such as clinical observation of clonic movements in the isolated forearm and recording of the electromyogram or electroencephalogram. We used a clinical observation of clonic movements in the isolated limb using the stopwatch. The study would have been strengthened if cerebral seizure activity had been measured directly by electroencephalography and electromyography, but this was not available routinely in our hospital. The complex psychomotor tests were not used for recovery assessment in our study.

**Conclusion**

From the current study, we conclude that there are individual advantages of the two induction agents. Propofol has the advantage of having shorter induction time and rapid and smooth recovery as compared to etomidate. It can be especially useful in elderly patients since it provides a more rapid recovery of cognitive function. Etomidate has the advantage of a longer seizure duration and stable hemodynamics. It may be indicated when seizures are too short and possibly subtherapeutic despite maximum stimuli. It can be a useful alternative in patients achieving suboptimal therapeutic responses to ECT. Hence, we conclude that etomidate is a better induction agent as compared to propofol in modified ECT.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

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