Section: Anesthesiology

Original Research Article

COMPARISON OF ETOMIDATE VERSUS PROPOFOL AS AN INDUCTION AGENT FOR MODIFIED ELECTROCONVULSIVE THERAPY

Aiswariya. D[1], Lalitha. M[2], Sabarish. S[3], Asmita. V.C[4]

[1]Post graduate, Department of Anesthesiology, Dhanalakshmi Srinivasan Medical College and Hospital, Perambalur, India
[2]Professor and Head, Department of Anesthesiology, Dhanalakshmi Srinivasan Medical College and Hospital, Perambalur, India
[3]Consultant, Department of Intensive Care, GKNM Hospital Coimbatore, India.
[4]Assistant Professor, Department of Anaesthesiology, Dhanalakshmi Srinivasan Medical College and Hospital, Perambalur India.

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Corresponding Author:
Dr. Asmita.V.C,
Email: vcasmita@gmail.com.

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Abstract

Background: Electroconvulsive therapy has improved safety and effectiveness over the past four decades, and an ideal anaesthetic should provide smooth, rapid initiation, rapid recovery, and attenuation of seizure activity. Etomidate and propofol were compared regarding the effectiveness of seizure and haemodynamic changes. The study aimed to compare the effect of etomidate versus propofol as an induction agent for modified electroconvulsive therapy. **Materials and Methods:** This observational study was conducted at the Department of Anaesthesiology and critical care, Dhanalakshmi Srinivasan Medical College and Hospital, Siruvachur, for one year (March 2021-March 2022). Sixty patients were divided into Group A and Group B. Group A received Inj. Propofol 1mg/kg body weight IV with 1ml of 2% lignocaine hydrochloride as the anaesthetic agent, and Group B received Inj. Etomidate 0.2mg/kg body weight IV. **Results:** Among 60 patients, 60% were male,40% were female, and most participants were between 31 and 35 years (41%). No significant difference in mean weight between groups. ASA Grade I (65%) patients, ASA Grade II (35%) patients, 12 patients are BPAD with mania, 13 patients with recurrent depressive disorder,and 25% of patients diagnosed with Schizophrenia. The mean heart rate and mean arterial pressure increased steadily after induction and relaxation, with a statistically significant difference in the T-test for Induction and ECT. Group A had significantly shorter seizure and recovery times than Group B. **Conclusion:** Etomidate produces better seizure quality than propofol, while propofol reduces seizure duration. Etomidate could be a useful alternative for patients with inadequate seizure duration.

INTRODUCTION

Electroconvulsive therapy (ECT) is a treatment that has been controversial since its introduction in 1938 but is effective in the treatment of mood disorders and schizophrenia. Changes in ECT practice over the past four decades have improved its safety and effectiveness. In 1934, pentylenetetrazol (Metrazol) was used to induce epileptic fits, insulin (Sakee) and Hexafluorodiethy.[1] ether were used to modify drug-induced convulsions, gallamine and succinylcholine were used to modify seizure activity, and intravenous short-acting barbiturates and depolarizing muscle relaxants were used to produce modified electroconvulsive therapy.[1,2] Electroconvulsive therapy (ECT) uses an electrical current to create a generalized cerebral seizure. An ideal anaesthetic for ECT should provide smooth, rapid initiation, rapid recovery, and attenuation of the physiological effects of seizure activity. Etomidate and propofol have been used as inducing agents for modified ECT.[3,4] Although it is primarily used to treat patients with major depression, patients with schizophrenia, schizoaffective disorder, catatonia, neuroleptic malignant syndrome, and bipolar disorder may also benefit. However, the practice carries a stigma due to misinformation about the methodology of the procedure.[5]

The antidepressant effect sets in relatively quickly and can last up to a few years. Overall, the mortality rate with controlled administration of ECT is very low but leads to mild memory loss in the long term. ECT is often used in pregnant and elderly patients to

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prevent side effects of psychotropic drugs. Although its mechanism of action is multifactorial, ECT causes changes in cerebral blood flow and regional metabolism.[6] Improving Healthcare Team Outcomes Today, ECT is widely used to treat a variety of mental health disorders in addition to depression. The benefits of ECT become visible after several sessions, and the results are permanent. The key is to educate the patient and their family about ECT, as the procedure has been associated with many false and illogical beliefs. The antidepressant effect sets in relatively quickly and can last up to a few years. Although its mechanism of action is multifactorial, ECT causes changes in cerebral blood flow and regional metabolism. Most patients who undergo ECT have a positive response without any adverse consequences.[7]

Etomidate is an anaesthetic with minimal changes in blood pressure and heart rate. It is useful for general anaesthesia in cardiac surgery and patients with poor cardiac function. It also offers benefits for the induction of haemorrhagic shock. Etomidate increases the latency and decreases the amplitude of auditory evoked potentials, increases the latency and decreases the amplitude of somatosensory evoked potential amplitudes and is less suppressed than other anaesthetics.[8,9] Etomidate has been linked to postoperative nausea and vomiting, similar to that after barbiturates and higher than that after propofol. Adrenal toxicity, sepsis and exogenous steroids have been reported, but the effect of etomidate on clinical outcomes has not been studied in a large population.[10] Therefore, the study aimed to compare the effect of etomidate versus propofol as an induction agent for modified electroconvulsive therapy.

MATERIALS AND METHODS

This observational study was conducted at the Department of Anaesthesiology and critical care, Dhanalakshmi Srinivasan Medical College and Hospital, Siruvachur, for one year (March 2021-March 2022). The ethical approval was obtained from the Institutional ethics committee of Human Subjects (ISCHS), Dhanalakshmi Srinivasan Medical College and Hospital. The technique was explained, and informed consent from the patients was obtained.

Inclusion Criteria

Patients aged 18-60 years with ASA grades I and II of both genders were included.

Exclusion Criteria

Patients under <18 years and >60 years, pregnant women, patients with ASA grades III and IV, and patients with epilepsy and neuromuscular disorder were excluded.

Sixty patients were divided into Group A and Group B. Group A received Inj. Propofol 1mg/kg body weight IV with 1ml of 2% lignocaine hydrochloride as the anaesthetic agent, and Group B received Inj. Etomidate 0.2mg/kg bodyweight IV. Duration of seizure, rate, blood pressure, SPO2, and recovery outcome from anaesthesia was noted before, during, and after ECT. Patients were kept NPO for 8 hours before the procedure.

Pre-anaesthetic medications with sedatives or narcotics were not required and may only prolong the anaesthetic recovery time. Reassurance from the Psychiatric staff should be sufficient to allay the patient's fear. All patients had undergone a pre-anaesthetic checkup one day before the surgery. Patients were evaluated for the presence of any systemic disease, and Boyle's machine was checked. Appropriate size endotracheal tube, working laryngoscope- size 3 and 4 Macintosh and McCoy blades, stylet, bougie, and working suction apparatus was kept ready before the procedure. Emergency drug trays of atropine, adrenaline, ephedrine, and dopamine were kept ready. Patients were shifted to the operation theatre and connected to the standard multimonitor, monitoring the ECG, SpO2, non-invasive automated blood pressure, and heart rate were recorded. Intravenous access was secured using an 18G or 16G IV cannula.

All patients were injected with Inj. Glycopyrrolate 0.2mg IV. Then, the patient was pre-oxygenated for 3 mins with 100% oxygen and induced with an IV anaesthetic agent as they were allocated till loss of verbal contact. A tourniquet applied to the left arm was inflated to isolate the limb to permit accurate measurement of motor seizure. After checking able to ventilate, muscle relaxant succinylcholine 0.5mg/kg IV was administered to all patients. Once fasciculations subsided, a bite block was inserted to prevent tongue biting. The psychiatrist was allowed to place bitemporal electrodes on the forehead. A brief stimulus was given to produce seizures. Seizure duration in isolated limbs was noted. Subsequently, the patient was ventilated with 100% oxygen until the return of spontaneous respiration. Once patients responded to the command with eye-opening, they were shifted to the post-anaesthesia care unit for further follow-up.

Statistical Analysis

Data were entered into MS Excel, and analysis was done using SPSS. The description of studyvariables was expressed using Mean and Standard deviation. Two-tailed independent sampleT-test was used to compare the variables. A p-value <0.05 was considered significant.

RESULTS

Among 60 patients, 60% were male, and 40% were female. Most participants between 31- 35 years, around 41%, between 20-25 years, were20%, 27% between 26- 30 years and remaining more than 36 years of age.

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Table 1:

		Frequency	Percentage
Gender	Male	36	60
	Female	24	40
Age group	20-25	12	20
	26-30	16	26.7
	31-35	25	41.7
	36-40	2	3.3
	41-60	5	8.3
ASA Grade	I	39	65
	II	21	35
Diagnosis	BPAD with depression	10	16.6
	BPAD with mania	12	20
	Recurrent depressive disorder	13	21.6
	Schizophrenia	25	41.7

ASA Grade I (65%) patients and ASA Grade II (35%) patients.10 patients are with a diagnosis of BPAD with depression,12 patients are BPAD with mania, 13 patients with recurrent depressive disorder, and 25% of patients with a diagnosis of Schizophrenia (Table 1).

Table 2: Mean weight, seizure and recovery time between groups

Variable	Group A	Group B	P value
Weight	61.08 ± 11.95	65.3 ± 10.98	0.342
Seizure time	41 ± 14.3	56 ± 4.3	0.005
Recovery time	6.7 ± 0.45	7.8 ± 4.45	0.004

The mean weight in Group A was 61.08 ±11.95, and Group B was 65.3 ±10.98. There is no significant difference in the mean weights of the two groups (Table 2).

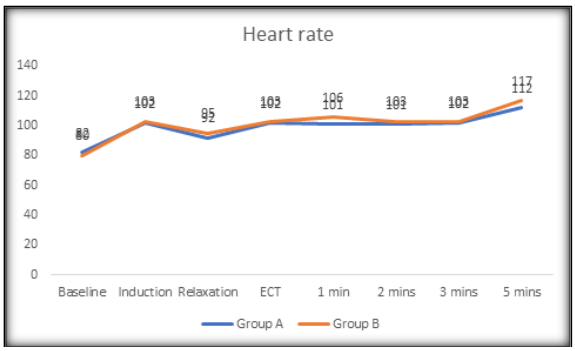

Figure 1: Heart rate between groups

Mean heart rate in both groups, once induction and muscle relaxant was given, dropped and steadily increased during the ECT procedure and increased in 1, 2, 3 and 5 minutes. It was noticed that there was no significant difference in mean values of the heart rate in 2 groups (Figure 1).

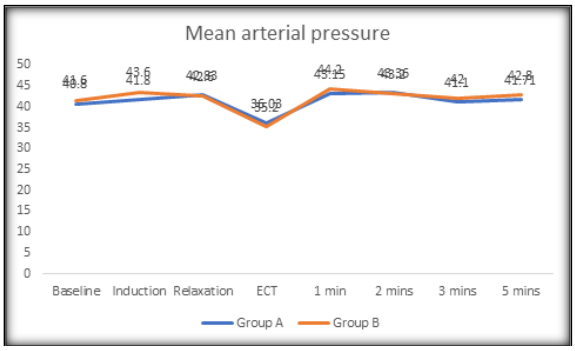

Figure 2: Mean arterial pressure between groups

Mean Arterial pressure showed a steady increase from baseline after induction and relaxation; during ECT increased at 1, 2, 3 and 5 mins. It was noticed that there was a statistically significant difference in the T-test for Induction and ECT procedure when mean arterial pressure was compared (Figure 2).

In Group A, the mean seizure time was 41 seconds, and in Group B, the mean seizure time was 56 seconds, and there was a statistically significant difference between the two groups. The mean recovery time in Group A was 6.7 Seconds, and in Group B was7.8, and there was a statistically significant difference between the two groups (Table 2).

DISCUSSION

ECT is the mainstay of treatment for psychiatric disorders not amenable to standard pharmacological treatment. Usually performed under general anaesthesia, the choice of an ideal anaesthetic has always been controversial, given the different pharmacodynamics of commonly used intravenous agents. Although several studies have been conducted to compare the effects of thiopentone and propofol on hemodynamic parameters and seizure duration during modified ECT, etomidate has been used sparingly. However, the availability and resurgence of etomidate in anaesthesia prompted us to compare and evaluate its effect on hemodynamic variables and motor seizure duration compared to propofol during anaesthesia for ECT.A literature review found that most studies on ECT measured HR and blood pressure before and after the seizure, ignoring the peak cardiovascular changes that occur during the seizure. In addition, not all studies reach similar conclusions regarding the hemodynamic

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profile and are limited by either the small sample size or the retrospective nature of the studies.[11-13]

In our study, the higher HR and MAP during seizure activity and up to 3 minutes after the seizure only in the etomidate group than in the propofol group can be explained by the cardiovascular depressant of propofol. The effects of propofol are dominated by the sympathetic stimulation induced by a seizure-induced during ECT. Although absolute MAP values were lower in the propofol group after induction at all-time intervals; but, this change from respective baseline values wasinsignificant, suggesting that although the propofol-induced decrease in hemodynamics was greater compared to etomidate, it was still not significant to cause potential adverse effects. A similar hemodynamic profile after administration of etomidate at 3 and 5 minutes has been observed in other studies. However, previous studies did not observe an increase in heart rate or increase from baseline at 1-minute post-attack. This discrepancy relative to our study can be explained by the time lag between the administration of inducing agents and the time at which seizure activity (ECT) was initiated. This delay may be less in previous studies than ours.

The results of the present study conflict with a study by Rosa et al.[11] who showed no significant difference in HR from baseline in both the etomidate and propofol groups immediately after the seizure. This can be illustrated by using a comparatively higher dose of etomidate (0.15-0.30 mg/kg) and propofol (1.5 mg/kg) in their study compared to relatively lower doses of 0.2 and 1 mg/kg etomidate and propofol, respectively in our study. MAP increased in the etomidate and propofol groups during and after the attack. Absolute MAP values and the change in these values from baseline were significantly greater in the etomidate group during and 1 minute after. Similar trends were observed for SBP and DBP. However, it may be appropriate to add that we only considered MAP values as this is more important for hemodynamic stability and monitoring during anaesthesia.

Although etomidate is considered a cardio-stable agent in routine anaesthetic practice due to the lack of hypotension during induction, the increase can be explained by the fact that patients were not administered any premedication or inhalation that could presumably attenuate sympathetic stimulation. The increase in MAP and HR from baseline observed in our study in etomidate compared to the propofol group is consistent with previous studies. Therefore, it is safe to conclude from these observations that propofol is more effective in dampening the sympathetic response to seizures and providing more cardioprotection. However, the conclusion regarding the hemodynamic profile contradicts our initial hypothesis. In contrast, some studies show a significant decrease with etomidate or no difference in HR or MAP when using either inducer. This difference may be due to the different methodology, small sample size, or use of variable doses of propofol or etomidate.

Most studies comparing EEG and EMG seizure duration with these two drugs have observed longer seizure duration using etomidate. In addition, decreased seizure duration with increasing propofol dose has previously been reported by other investigators. The comparatively longer seizure duration in both groups reported by Avramov et al. compared to the present study could reflect the monitoring methods used to measure.[14] Their study results showed that the duration of EEG and motor seizures were longest after etomidate and shortest after propofol. All hemodynamic parameters (HR, SBP, DBP and MAP) were less elevated in group P compared to group E after ECT at all study time intervals.

In our study, propofol appeared superior to etomidate in attenuating the cardiovascular stress response to ECT with minimal hemodynamic changes. Similar results were reported in a study by Gazdag et al. that compared propofol and etomidate for ECT in patients with schizophrenia. Their results showed that the increase in MAP was significantly less when using propofol than when using etomidate (8.1 ± 10.2 mm Hg, 18.3 ± 11.2 mm Hg, P=0.001).[15] Zgola et al. also found similar results with propofol and etomidate in patients undergoing implantable cardioverter defibrillator trials. Their study results showed that propofol significantly reduced the values of all measured hemodynamic parameters.[16] In our study, patients in the propofol group achieved consciousness earlier after induction than patients in the etomidate group.

CONCLUSION

The study concluded that etomidate was found to produce better seizure quality, while propofol resulted in a reduction in seizure duration. Propofol showed good hypnosis, with its stable induction and rapid recovery time compared to etomidate. Hence, etomidate could be a useful alternative to propofol in patients with inadequate seizure duration.

Limitations

Fewer sample size was one of the main limitations of the study. Also, this was a single-centric study, so a further multi-centric study with increased sample size is recommended.

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