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Original Article

Anaesthesia Section

Effect of Intravenous Etomidate versus Propofol on Seizure duration and Haemodynamic Response during Modified Electroconvulsive Therapy: A Randomised Clinical Trial

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ABSTRACT

Introduction: Seizure duration in Electro convulsive Therapy (ECT) is a clinically important factor in managing psychiatric patients subjected to the procedure. Various induction agents are being used with varying efficacy on seizure duration and haemodynamic response like thiopentone, propofol, ketamine and etomidate. As repeated ECT sessions pose significant risk of general anaesthesia and its complications, in this study the efficacy of etomidate and propofol was compared.

Aim: To assess the seizure duration and compare haemodynamic variables using etomidate and propofol.

Materials and Methods: A randomised clinical trial was done in the Department of Anaesthesiology, at tertiary care hospital between July 2021 to June 2022 on 40 patients of age group 18 to 60 years of either sex, belonging to American Society of Anaesthesiologists (ASA) Grade-I and II scheduled for ECT. Patients were allocated into two groups. Preinduction baseline values of Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) were recorded using pulse oximeter and Non Invasive Blood Pressure (NIBP). Patients were induced with either, inj. etomidate 0.2 mg/kg (Group E) or inj. propofol 1 mg/kg (Group P). HR, SBP, DBP and MAP were recorded soon after induction, after application of stimulus and at one minute interval after electric 156 shock for five minutes and then at five minutes interval. Statistical analysis was done by using the unpaired Student's t-test for quantitative data. The p-value <0.05 was considered significant.

Results: Demographic data were similar in both the groups. The mean seizure duration in Group E (51.25±9.01 seconds) was greater than in Group P (38.30±9.92 seconds) and was statistically significant. There was significant increase in the HR in both groups and the HR did not reach the baseline even after 10 minutes. There was a rise in the mean SBP by approximately 7 mm Hg in the Group P compared to 4 mm Hg in the Group E. The mean DBP rise in the propofol group was 6 mm Hg as compared to 5 mm Hg in the Group E. The MAP in both the groups increased by 7 mm Hg. The parameters reached the baseline earlier with Group P compared to Group E.

Conclusion: The study concludes etomidate has a distinct advantage over propofol in producing seizures of adequate duration during ECT whereas propofol blunts the sympathetic response to ECT more effectively than etomidate.

Keywords: Cardiovascular effects, Psychiatry patient, Sympathetic response

INTRODUCTION

The ECT, also known as electroshock, is a well-established, albeit controversial psychiatric treatment in which seizures are electrically induced in anaesthetised patients for therapeutic effect. ECT is most often used as a treatment for severe major depressive disorder which has not responded to other treatment [1], and is also used in the treatment of mania (often in bipolar disorder), catatonia and schizophrenia. For the safe conduct of ECT, an effort to avoid or minimise the physiologic sequelae and the attendant complications of ECT, a technique of modified ECT has evolved gradually, featuring use of muscle relaxation and induction agents without the concomitant abolition of the beneficial effects [2]. The commonly used muscle relaxant is a short acting depolarising agent succinylcholine. Various induction agents were tried viz., diazepam, ketamine, methohexitone, sodium thiopentone. However, the attendant cardiovascular effects are inadequately attenuated with its use [3]. The search for an ideal anaesthetic agent for ECT has been an ongoing process. Most of the anaesthetic agents used possess anticonvulsant properties because of their effects on the Gamma-Aminobutyric Acid (GABA) receptors.

Following application of the electrical stimulus during ECT, there is a vagally mediated short lived bradycardia which is replaced by a sympathetically mediated tachycardia and rise in blood pressure [4]. Accordingly there is a sharp rise in the plasma catecholamine levels. This produces a short lived sharp increase in myocardial workload which may pose significant risk for patients with coronary artery disease and congestive cardiac failure. Hence, use of agents which would attenuate this adverse physiologic consequence would be preferred.

Etomidate, a imidazole derivative is a short acting intravenous anaesthetic agent used for the induction of general anaesthesia. It acts by inhibiting the reticular activating system and mimics action of GABA inhibition. The R (+) isomer of Etomidate particularly appears to bind specifically to a subunit of the GABAA receptor, thus increasing the affinity for inhibitory neurotransmitter i.e., GABA [5].

Propofol structure includes phenol ring substituted with two groups of isopropyl (2, 6-diisopropylphenol) is primarily a hypnotic agent and has rapid onset of action for induction of anaesthesia. Propofol allosterically increases affinity of binding GABA to GABAA receptor. This receptor is coupled to a chloride (Cl^-) channel and activation of the receptor results in hyperpolarisation of the nerve membrane. Propofol binds to multiple ion channels and receptors. The α^2-adrenoceptor system indirectly causes sedation due to propofol. Propofol also causes inhibition of NMDA (N-methyl D-aspartate) receptor, which is a subtype of glutamate receptor [6].

Administration of etomidate in ECT has variable impact on seizure duration and haemodynamic parameters, when compared to propofol. Improved seizure duration observed with etomidate versus propofol has been reported in various studies. However, some studies have shown that etomidate has better haemodynamic profile in patients undergoing modified ECT. Jindal S et al., in their study concluded that among haemodynamic parameters, there was a significant increase in HR and significant fall in MAP after induction with propofol as compared to etomidate. Hence, stating etomidate has stable haemodynamics compared to propofol [7]. Another study conducted by Mansuri Y and Dave J, showed that etomidate has stable haemodynamics compared to propofol during modified ECT [8]. This study was undertaken to study the effect of propofol in suppressing the sympathetic response induced by modified ECT, thus enhance safety in patients undergoing ECT. Thus, the present study aimed to compare the effect of propofol and etomidate on seizure duration and haemodynamic response when administered during ECT.

The primary and secondary measure of this study was to assess the seizure duration and to compare haemodynamic response of etomidate and propofol respectively.

MATERIALS AND METHODS

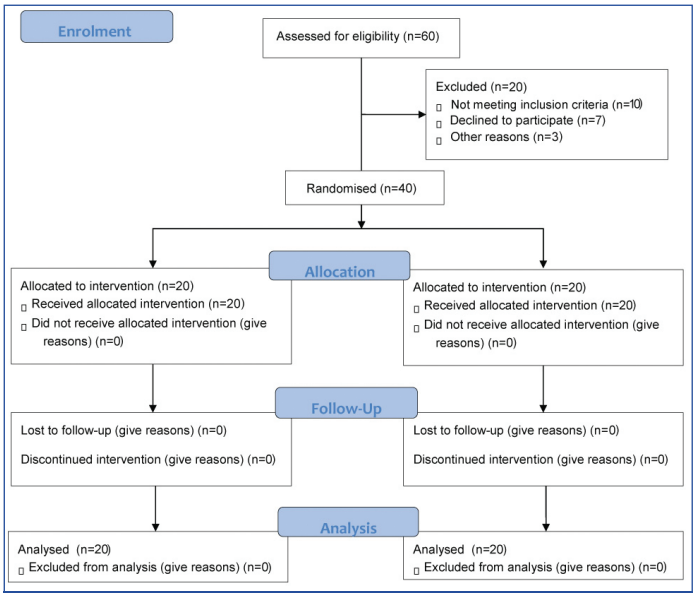
A randomised double-blinded, clinical study was conducted in the Department of Anaesthesiology, at tertiary care hospital between July 2021 to June 2022 for the period of one year after approval from Institutional Ethical Committee (IEC) (MDC/DOME/2158).

Inclusion criteria: A total of 40 patients between the age group of 18-60 years belonging to ASA grade I and II, taking ECT for first time, of either gender and with no absolute contraindication to ECT were included in the study. Informed written consent was obtained from the patient`s close relative before being included in the study.

Exclusion criteria:

1. Pregnant women.
2. Hypertensive patients
3. HR less than 60 beats/minute.
4. History of allergy to any drug.

Sampling was done by simple random sampling using computer generated table. Consolidated Standards of Reporting Trials (CONSORT) flow diagram is given in [Table/Fig-1].



[Table/Fig-1]: CONSORT flow diagram.
n: Number of subjects assessed

Sample size was calculated using Open-epi software (AG Dean, KM Sullivan, MM Soe–3.03/ September 22, 2014) considering 95% confidence interval, 90% power of study.

Sample size calculation formula:

$$n=\frac{(Z\alpha + Z\beta)2*2*(S)^2}{d^2}$$

Where, Zα: Two tailed significance level 5% = 1.96

Zβ: Power of study 90% = 0.94

(Zα + Zβ) 2= (1.96 + 0.94)2= 8.4

S= 16.4 (S= standard deviation of SBP, d = mean difference of SBP) [9]

d=15

$$n=\frac{8.4\times2\times(16.4\times16.4)}{(15\times15)}$$

=20 in each group

The sample size obtained was 20 in each group.

Procedure

A thorough preanaesthetic evaluation was conducted a day prior to ECT and routine investigations like Haemoglobin%, urine routine, blood sugars, blood urea, serum creatinine, ECG, Chest X-ray were carried out for all patients. On the day of ECT, each patient`s investigations verified and were found to be within normal limits. Antipsychotic drugs were omitted on the day of ECT. Overnight fasting of eight hours before the procedure was confirmed. Patients were randomly allocated using computer generated random numbers into two groups of 20 patients each.

Anaesthetic machine and all equipments were checked and kept ready along with the crash cart. On arrival of the patient to the ECT room, ECG, pulse oxymeter and NIBP monitors were attached and baseline HR, SBP, DBP and MAP were recorded using a NIBP monitor (Larsen and Turbo model star 50). An intravenous line was secured on the dorsum of left hand using a 20 G intravenous cannula. All patients were premedicated with Inj. glycopyrrolate 0.2 mg i.v. and preoxygenated for three minutes. Induction was done using etomidate (0.2 mg/kg) or propofol (1 mg/kg) depending on the group allocated by an anaesthesiologist who was blinded for the study.

Group E (Etomidate): Patients in this group received etomidate (0.2 mg/kg) slowly over 15 seconds. Induction was confirmed by loss of eyelash reflex.

Group P (Propofol): Patients in this group received propofol (1 mg/kg) slowly over 15 seconds.

After noticing of loss of eyelash reflex, blood pressure cuff applied to the lower limb was inflated to isolate the foot and permit accurate measurement of motor seizure duration. After confirming, the patient could be ventilated. Injection succinylcholine 1.0 mg/kg was given for muscle relaxation. Patients were ventilated with 100% O_2 until fasciculations subsided.

As soon as the patient was relaxed, a mouth gag was inserted and a bitemporal ECT was administered by the psychiatrist. Motor seizure duration was noted again by the anaesthesiologist who was blinded for the study and was different from the anaesthesiologist who allocated the groups. The mouth gag was changed to Guedel airway after the seizure subsided and ventilation was assisted with the face mask and 100% oxygen until return of spontaneous respiration. The patient was observed for 10 minutes in the ECT room and later was monitored in the recovery room for an hour.

The seizure duration is defined as time interval between administration of electrical stimulus and loss of visible fasciculation's in the isolated limb [7].

Monitoring: Seizure duration was noted in all patients. All patients had continuous pulse oximeter, ECG monitoring and systolic, diastolic and MAP were recorded and monitored using an automated blood pressure machine set to record every minute.

Baseline HR, SBP, DBP and MAP were noted just before securing the intravenous cannula. The same parameters were noted after loss of eyelash reflex following induction, immediately after seizure cessation following delivery of the electric shock and at 1 minute interval for 5 minutes and once after five minutes (10 minutes post ECT).

STATISTICAL ANALYSIS

Data are presented as mean and standard deviation. Statistical analysis was done by using the unpaired Student's t-test for quantitative data. Comparison of proportions (percentage) of the two groups was done using test for proportions was done using data analysis and $p<0.05$ was considered significant.

RESULTS

In this comparative study, 40 patients (20 in each group) undergoing ECT were randomly selected. Both the groups were comparable with respect to age, sex, weight and diagnosis. [Table/Fig-2] shows mean age in Group E was 28.6 years and 34.25 years in Group P and weight in Group E was 48.90 kg and 51.40 kg in Group P which are not statistically significant. Male to female proportion was similar.

Variables	Group E (Mean±SD)	Group P (Mean±SD)	p-value
Age (years)	28.6±10.56	34.25±14.02	0.1582
Weight (kg)	48.90±5.63	51.40±12.60	0.4229
Gender n (%)			
Male	10 (50%)	11 (55%)	0.9523
Female	10 (50%)	9 (45%)	0.9473

[Table/Fig-2]: Mean age, weight and gender in the two groups.

[Table/Fig-3] depicts the percentage of distribution of diagnosis of disease in the two groups, with a varying pattern of distribution. Majority of the patients receiving ECT were subjects diagnosed as schizophrenia followed by psychosis and depression.

Diagnosis	Group E n (%)	Group P n (%)	p-value
Schizophrenia	10 (50)	8 (40)	0.8896
Depression	3 (15)	3 (15)	1.0000
Catatonia	3 (15)	2 (10)	0.8054
Mania	2 (10)	3 (15)	0.8054
Psychosis	2 (10)	4 (20)	0.6914

[Table/Fig-3]: Distribution of diagnosis in the two groups.

[Table/Fig-4] shows the mean seizure duration in Group E which was greater than in Group P and was statistically significant.

	Group E (Mean±SD)	Group P (Mean±SD)	p-value
Seizure duration (in seconds)	51.25±9.01	38.30±9.92	0.00011

[Table/Fig-4]: Mean seizure duration between the groups.

[Table/Fig-5] depicts that the HR in the both groups significantly increased after application of electric shock, the increase continued for three minutes after the electric shock but does not reach the baseline even at 10 minutes. The HR trend in the Group E and that in the Group P were significantly different and the values immediately after applications of electric shock were significantly higher with the Group E compared with the Group P.

[Table/Fig-6] shows that the SBP in the Group E increased after application of electric shock and the increase continued until four minutes after the application of electric shock. In the Group P, there was an increase in the SBP until two minutes of application of electric shock. The increase was greater at two minutes of application of electric shock in the two groups.

Events	Group E	p-value (comparison with baseline)	Group P	p-value (comparison with baseline)	(Group E versus Group P) p-value
Preinduction	84.05±13.65		78.40±7.00		0.108
Postinduction	85.25±16.55	0.1632	83.55±12.12	0.0060	0.712
Postshock	93.35±20.63	0.0054	85.45±12.36	0.0014	0.150
1 min	96.70±24.31	0.0014	89.30±16.21	0.0027	0.264
2 min	97.70±25.49	0.0021	87.20±12.16	0.0005	0.104
3 min	94.00±20.14	0.0013	84.85±11.06	0.0022	0.082
4 min	97.70±25.36	0.0067	84.55±9.21	0.0012	0.035
5 min	97.00±23.91	0.0038	83.20±8.22	0.0025	0.019
10 min	93.60±21.59	0.0147	82.95±7.78	0.0031	0.044

[Table/Fig-5]: Comparison of Heart Rate (HR) in the two groups with the baseline.
The significance level, or p-value, is calculated using the t-test.
When the p-value is less than 0.05 ($p<0.05$), the conclusion is that the two means are statistically significant and p-value less than 0.001 ($p<0.001$) as statistically highly significant

Events	Group E	p-value	Group P	p-value	Group E versus Group P p-value
Preinduction	124.35±13.08		124.60±17.77		0.9599
Postinduction	124.80±11.01	0.3836	121.20±17.14	0.0412	0.4343
Postshock	128.50±12.83	0.0030	131.50±31.65	0.0222	0.6967
1 min	129.90±12.69	0.0146	130.65±22.49	0.0473	0.8973
2 min	131.50±13.65	0.0103	132.70±24.81	0.4377	08507
3 min	129.55±11.55	0.0073	124.75±20.10	0.4844	0.3603
4 min	127.25±13.43	0.0423	124.80±20.25	0.4788	0.6546
5 min	125.35±12.24	0.1919	122.75±18.97	0.3106	0.6096
10 min	124.90±12.15	0.2901	121.80±18.23	0.2138	0.5306

[Table/Fig-6]: Comparison of Systolic Blood Pressure (SBP) in the two groups with the baseline.
The significance level, or p-value, is calculated using the t-test.
When the p-value is less than 0.05 ($P<0.05$), the conclusion is that the two means are statistically significant and p-value less than 0.001 ($P<0.001$) as statistically highly significant

[Table/Fig-7] depicts that the DBP in the Group E increased after application of electric shock and the increase continued until four minutes after the application of electric shock. In the Group P, there was an increase in the DBP until two minutes of application of electric shock.

Events	Group E	p-value	Group P	p-value	Group E versus Group P p-value
Preinduction	76.40±7.21		80.35±13.46		0.2545
Postinduction	78.05±7.56	0.1385	77.20±14.11	0.0248	0.8136
Postshock	81.00±9.51	0.0106	86.25±24.74	0.0483	0.3813
1 min	82.50±10.50	0.0031	85.90±22.36	0.0465	0.5419
2 min	79.55±6.87	0.0354	87.20±21.24	0.0293	0.1336
3 min	79.55±5.38	0.0407	76.70±18.25	0.1251	0.5070
4 min	79.10±5.41	0.0414	75.65±16.55	0.0591	0.3812
5 min	75.40±8.00	0.2744	75.35±17.92	0.0570	0.9910
10 min	74.20±6.41	0.0629	75.65±15.16	0.0651	0.6958

[Table/Fig-7]: Comparison of Diastolic Blood Pressure (DBP) in the two groups with the baseline.
The significance level, or p-value, is calculated using the t-test.
When the p-value is less than 0.05 ($P<0.05$), the conclusion is that the two means are statistically significant and p-value less than 0.001($P<0.001$) as statistically highly significant

[Table/Fig-8] shows that the MAP in the Group E increased after application of electric shock and the increase continued until four minutes after the application of electric shock. In the Group P, there was an increase in the MAP until two minutes of application of electric shock. The trends of the MAP were

significantly increased in the Group E compared with the Group P.

Events	Group E	p-value	Group P	p-value	Group E versus Group P p-value
Preinduction	91.65±12.95		92.60±15.45		0.8342
Postinduction	93.30±9.00	0.2148	90.15±13.77	0.0429	0.3972
Postshock	98.05±11.28	0.0035	99.00± 20.78	0.0383	0.8436
1 min	99.30±9.27	0.0005	98.30±22.23	0.0333	0.8537
2 min	98.80 ±9.02	0.0007	100.55 ±25.79	0.0400	0.7761
3 min	96.30±8.83	0.0488	90.45±19.73	0.2680	0.2337
4 min	94.55±10.44	0.0472	90.95±18.92	0.3287	0.4608
5 min	91.60±10.00	0.4880	88.40±19.36	0.1231	0.5153
10 min	91.35±9.69	0.4287	87.20±18.21	0.0587	0.3738

[Table/Fig-8]: Comparison of Mean Arterial Pressure (MAP) in the two groups with the baseline.
The significance level, or p-value, is calculated using the t-test.
When the p-value is less than 0.05 (P<0.05), the conclusion is that the two means are statistically significant and p-value less than 0.001(P<0.001) as statistically highly significant

With regards to the complications observed during the study the incidence of pain on injection was 25% in the Group P and none had pain in the Group E. There were no complaints of pain on injection, myoclonus or increased tonus-related complications.

DISCUSSION

The ECT has a well-established role in the management of patients who have not responded to psycho-pharmacological treatment [10]. The procedure itself consists of programmed electrical stimulation of the central nervous system to initiate seizure activity. In terms of haemodynamic effects, seizure activity causes an initial parasympathetic discharge, later followed by sympathetic discharge. In terms of patient comfort and amnesia during the operation, anaesthesia has elevated ECT to a new level with the introduction of intravenous anaesthetic drugs, neuromuscular blockade, and assisted or controlled ventilation with 100% oxygen in 1963 [10]. The ideal ECT induction drug would guarantee quick unconsciousness, be painless upon injection, have no haemodynamic side-effects, not change seizure duration or amplitude, and be reasonably priced. [11]. The present study has compared the seizure duration and haemodynamic responses to ECT with etomidate and propofol as induction agents. The results of the present study indicate that there are differences in seizure duration and the haemodynamic responses on induction with these two agents.

In the present study, the mean seizure duration was significantly prolonged in etomidate group (51.25±9.01 seconds) when compared to propofol group (38.30±9.92 seconds). This is attributed to the lack of anticonvulsant properties of etomidate which makes it preferred induction agent as it provides better therapeutic efficacy due to seizure prolongation. The seizure duration is defined as time interval between administration of electrical stimulus and loss of visible fasciculation's in the isolated limb. The aim of ECT is to obtain generalised convulsions over 20 seconds. Although there is no beneficial effect with only one seizure, clinical improvement can be observed with a total seizure time over 210 seconds. In the present study, in terms of SBP, DBP and MAP remained elevated upto four minutes in etomidate group compared to propofol group where the increase was only upto two minutes. The HR was considerably high in etomidate group compared to propofol group and did not reach the baseline in both the groups even after 10 minutes postshock. This can be explained by the cardiovascular depressant effect of propofol which dominates over the sympathetic stimulation caused by the seizure induced during ECT. Similar results were noted by Mehta D et al., in which propofol maintained stable haemodynamics during modified ECT, yet clinical applicability of etomidate outstripped propofol by a reasonable margin due to its better effect on seizure parameters. The increase in haemodynamic parameters in etomidate group here is explained by the increased apnoea time secondary to increased motor duration of the seizure and also its perceived effects on adrenocortical axis which were feared to be sustained than transient [12]. Shastry SB et al., compared propofol and etomidate on haemodynamic variables and seizure duration and concluded that seizure duration recorded was prolonged in a statistical significant manner whereas the scores used to assess haemodynamic parameters in both the groups were the same indicating similar efficacy in stabilising the haemodynamic changes [13]. This was attributed to the inherent advantage of blunting the autonomic response to induced seizures by the induction agents. Mansuri Y and Dave J, compared the effects between etomidate and propofol for anaesthesia during ECT in which they observed that duration of seizures was significantly prolonged in former group [8]. This can be explained by the propensity to prolong the seizure duration by etomidate.

A study carried out by Stadtland C et al., concluded a switch from propofol to etomidate during ECT course increases EEG and motor seizure duration [14], which was similar with the present study. Etomidate has the distinct advantage of producing seizures of adequate duration during ECT and should be used as first line measure in augmenting seizures in patients who have difficulties in inducing seizures by conventional method was the conclusion derived from a study carried out by Jensen KR et al., [15]. A retrospective study done by Patel AS et al., observed that patients who received propofol had longer courses of ECT and, consequently longer and costlier inpatients stays and concluded that etomidate could be an alternative induction agent [16]. Patil M et al., have studied neuroendocrinal responses in 60 patients undergoing ECT under propofol and thiopentone anaesthesia [17]. They found that subjects given propofol had significantly reduced Adrenocorticotropic hormone (ACTH) and cortisol responses compared to thiopentone. These humoral responses could have resulted in the lesser rise in the haemodynamic parameters in the propofol induced subjects. After application of the electric shock in the present study, there was a rise in the mean SBP of approximately 7 mm Hg (124.6 mm Hg to 131.50 mm Hg) in the propofol group. In comparison, in the etomidate group the rise was 4 mm Hg (124.8 mm Hg to 128.50 mm Hg). The mean DBP rise in the propofol group was 6 mm Hg (80.35 mm Hg to 86.25 mm Hg) as compared to 5 mm Hg (76.40 mm Hg to 81.00 mm Hg) in the etomidate group. The MAP in the both the groups increased by 7 mm of Hg. Although significant increase in mean blood pressure values were observed after ECT with both the agents, increase in blood pressure was slightly higher with propofol, when compared with etomidate but the values reached the baseline earlier i.e., by 2 minutes in Group P when compared four minutes in Group E following administration of electrical stimulus. Analysis of the data suggested that propofol was more effective in controlling haemodynamic response to ECT than etomidate. Also, it was found that the mean HR in both the groups increased from the baseline after application of electric shock. However the rise was lesser in the propofol group than the etomidate group. Propofol had the advantage of smooth induction, stable haemodynamic parameters, and rapid recovery as compared to etomidate. However, it was associated with shorter seizure duration. Etomidate had longer seizure duration which results in better clinical outcomes over propofol [11]. The results were similar to what was noted in the present study.

A similar comparative study by Jindal S et al., here apart from seizure duration, cognitive recovery profiles, haemodynamic parameters viz., HR and MAP were observed. Their observations were similar to the present study in which propofol was associated with a reduced acute haemodynamic responses

compared to etomidate [7]. Following induction with propofol, there was slight fall the systolic, diastolic and the mean blood pressure. This is attributed to its vasodilating property of propofol, which reduces the peripheral vascular resistance. In a study conducted by Lisanby SH, propofol and etomidate were compared during the ECT of patients with depression on the basis of their impact on seizure activity and on seizure induced haemodynamic reactions. When using propofol, the increase in MAP was significantly lower than when etomidate was used. They concluded that propofol was more effective in attenuating the seizure-induced increase in MAP than etomidate, and supported the use of propofol in patients with greater cardiovascular risk [18]. In a comparison study of thiopentone sodium, propofol, and etomidate for anaesthetic efficacy and effect on seizure duration in ECT, Vansola RH et al., found that propofol was more advantageous than etomidate and thiopentone due to stable haemodynamic boundaries, smooth enlistment and fast recuperation in contrast with etomidate and thiopentone. [19]. In a study on ECT by Rasmussen KG, has shown that after the electrical stimulus, there is a vagally mediated short lived bradycardia following sympathetically mediated tachycardia and rise in blood pressure [20]. The initial bradycardia was not noticed in any of the patient in our study. Premedication with intravenous glycopyrrolate could have aborted that phase in present study. The choice of anaesthetic for ECT depends on the anaesthetic needs to be met and agent's effect on the seizure threshold. Thus, the non barbiturate anaesthetic etomidate showed qualities to prolong the seizure. During the application of ECT, complications can occur at the induction and recovery stages. Present study did not encounter any complaints of pain on injection, myoclonus or increased tonus-related complications.

Limitation(s)

The study was conducted only in patients belonging to ASA Class-I and II physical status. Therefore, effects of propofol and etomidate for ECT in geriatric population and those having associated co-morbidities are yet to be studied. Since, the study was conducted with a sample size of 40 patients, 20 in each study group, results obtained cannot be generalised for entire population. Thus, further studies should be designed and conducted with larger study groups including patients with co-morbidities.

CONCLUSION(S)

Propofol has superior haemodynamics during ECT, yet the clinical utility of etomidate outpace it by a reasonable margin due to its improved effect on seizure parameters and the clinical conditions that require ECT. Hence, the present study inferes that etomidate can be a potential first line drug of choice in ECT in seizure resistant patients.

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