Section: Anaesthesiology

Original Research Article

A COMPARATIVE STUDY OF EFFECTIVENESS OF PROPOFOL, KETOFOL AND KETOFOL WITH DEXMEDETOMIDINE AS INDUCTION AGENTS FOR ELECTRO CONVULSIVE THERAPY

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ABSTRACT

Background: Thiopentone, propofol, ketamine and ketofol have all been used as induction agents for ECT. Dexmedetomidine is a potent alpha 2 agonist which has been tried recently as pre treatment along with ketofol as induction agent for ECT. In this study we compared propofol, ketofol and ketofol with Dexmedetomidine as inducing agent in ECT.

Materials and Methods: This prospective randomized double blind study was conducted inthirty patients between 18 and 65 yrs with ASA status I and II scheduled for ECT. All patients randomly received all study agents for first three sessions of ECT. The observations were compiled as Group P (Inj. Propofol 1mg/kg), Group K (Inj. Ketofol i.e. Inj. Propofol 0.5mg/kg + Inj. Ketamine 0.5mg/kg) and Group KD (Inj.Ketofol + Inj. Dexmedetomidine 0.5 mcg/Kg). Heart rate and mean blood pressure was recorded at pre op, 0, 5, 10 and 20 minutes after ECT. The seizure duration, time to spontaneous eye opening & obeying verbal commands, agitation score and post ECT myalgia scorewere recorded.

Results: We observed statistically significant difference in heart rate &mean arterial pressure post induction and at subsequent time interval where group K showed higher value than group P and group KD ($p < 0.05$). Group KD showed significantly longest seizure duration as well as time to spontaneous eye opening and time to obey verbal commands ($p=0.000$). It also showed a significantly lower agitation score & myalgia score compared to Propofol and Ketofolgroups ($p=0.000$).

Conclusion: Ketofol with dexmedetomidine combination appears to be superior in terms of better hemodynamic stability, increased seizure duration and less incidence of adverse effects although with slightly delayed recovery compared to ketofol and propofol. Therefore Ketofol with dexmedetomidine combination can be used as an effective & safe induction agent during ECT.

Keywords: Electroconvulsive therapy, Dexmedetomidine, ketamine, ketofol, propofol.

INTRODUCTION

Numerous evidence-based resources had highlighted electroconvulsive therapy (ECT) as a safe and efficacious treatment for various psychiatric disorders.[1,2] The purpose of ECT is to induce a controlled and monitored seizure of around 30 to 90 seconds duration.[1,3,4] ECT may be associated with haemodynamic changes such as hypotension and bradycardia followed by hypertension and tachycardia. After regaining consciousness post

ECT, patient may complain of confusion, agitation, headache and muscle stiffness.

The anaesthetic management for ECT involves use of an induction dose of intravenous anaesthetic followed by muscle relaxant. The goal is to get rapid loss of consciousness, avoidance of gross movement, minimal interference with seizure activity and reduction of hyper dynamic response.

Thiopentone, propofol, ketamine and recently ketofol have all been used as induction agents for ECT. Currently Propofol with its property of causing rapid smooth induction and recovery is the most commonly used induction agent in ECT. Its cardiovascular effect of hypotension is an added advantage in counteracting ECT induced hypertension. However dose dependent decrease in seizure duration is disadvantage of Propofol. Ketamine is also used as an induction agent for ECT. It shows increase in seizure duration and has antidepressant action. The drawback of ketamine is enhanced hemodynamic response, delayed recovery and increased risk of post ECT agitation. Ketofol(1:1 mixture of ketamine and propofol) is recently used as an induction agent in ECT. The cardiovascular properties of both propofol and ketamine balance each other in maintaining hemodynamic stability which is the advantage of ketofol.[5,6] Dexmedetomidine is a potent alpha 2 agonist having sedative, sympatholytic and analgesic effect without respiratory depression.[7] In ECT it is being tried recently as pretreatment along with ketofol as induction agent.

In this study we compared propofol, ketofol and ketofol with Dexmedetomidine as inducing agent in ECT. We hypothesized that all the three drug combinations are equally effective as induction agent in ECT.

MATERIALS AND METHODS

After ethical committee approval this prospective randomised control trial was conducted in 30 patients. To determine the sample size for our study, we utilized the online software Open Epi version 3 (https://www.openepi.com/SampleSize/SSCohort.htm), which accounted for this observed difference in seizure duration.[8] The calculated minimal sample size was 22 participants. However, in order to enhance the precision of our data, we decided to increase the sample size to 30 participants.

Patients of both sexes between the ages of 18 and 65 years with American Society of Anesthesiologists (ASA) physical status I and II scheduled for ECT were enrolled for the study.

Patients on chronic opiate use, pregnant females, and lactating mothers, patients with known allergy to the study drugs, patients with cardiovascular diseases and patients taking beta blockers were excluded from the study.

The primary outcome of our study was comparison of study drugs with respect to their effects on hemodynamic parameters, recovery parameters and seizure duration during ECT whereas incidence and severity of post ECT agitation and myalgia was secondary outcome.

All the patients were screened before enrolment in the study. After eliciting detailed history patients underwent complete medical and laboratory examinations. A voluntary written informed consent was taken from all the eligible and willing patients and their relatives. Before enrolment of first patient in this study; registration for clinical trial was done (CTRI/2023/04/051890).

Patients who undergo ECT at our hospital usually receive 6-8 sessions on alternate days according to the clinical response of the patient. First three sessions of ECT per patient were included in our study. Patients were randomized into three groups of 10 each using computer-generated random numbers. Allocation concealment was ensured using sequentially numbered, opaque, sealed envelopes.

Group A – received 10 ml NS followed by Inj. propofol (1mg/kg) for first session

Group B – received 10 ml NS followed by Inj. Ketofol (Inj. propfol 0.5mg/kg and Inj. ketamine 0.5mg/kg) for first session

Group C – received Inj. Dexmedetomidine 0.5 mcg/kg diluted up to 10 ml using NS followed by ketofol (Inj. ketamine 0.5 mg /kg and propofol 0.5mg/kg) for first session

Patients in group A received injection ketofol for second session and ketofol – dexmedetomidine combination in third session of ECT. Patients in group B received dexmedetomidine – ketofol for second session and propofol for third session of ECT. In group C, patients received Inj. Propofol in second session and ketofol in third session. All the three study drugs were used in each patient so as to avoid influence of patient and disease variant on the response of drug.

One anaesthesiologist prepared the study drugs while the second anaesthesiologist conducted the Anesthesia and observed the parameters. Patients, Outcome assessors and data analysts were blinded to group allocation.

Patients were kept nil per oral for 8 hrs prior to ECT. Weight of the patient was recorded. After arrival in the ECT room, an intravenous cannula of 20G was inserted into the arm and Ringer Lactate was started. The baseline parameters i.e. mean blood pressure (MAP), heart rate (HR) and oxygen saturation (SpO2) and ECG was recorded.

Inj. Glycopyrrolate (0.005 mg/kg) was given as premedication. MAP, HR and SpO2 were recorded two minutes after premeditation. The patients in ketofol and dexmedetomidine group received calculated dose of dexmedetomidine diluted in 10ml normal saline over 10 minutes. The patients in other two groups received 10ml NS. Then patients were induced with either inj. Propofol (1mg/kg) or ketofol (Inj. ketamine 0.5 mg /kg and propofol 0.5mg/kg) as per group allotted.

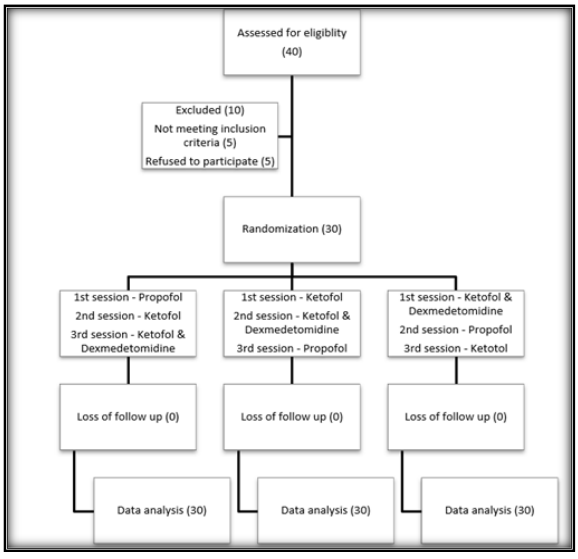
One of the lower limbs was isolated with sphygmomanometer cuff inflated to 100 mm of Hg above the systolic blood pressure to monitor the

duration of seizure activity. After isolating the limb, IV Succinylcholine in a dose of 1 mg/kg was administered and manual ventilation was performed with face mask and Bain's circuit using 100% oxygen at flow rate of 8L/min.

A bite block was used to protect the patient's teeth, lips and tongue. A supra threshold electrical stimulus was given via bi fronto temporal electrodes to induce seizure. The duration of the motor seizure, defined as the time from the application of shock to cessation of tonic clonic motor activity in the 'isolated' limb was recorded. Ventilation was assisted with oxygen throughout the procedure. MAP, HR, and SpO2were recorded at 0, 5, 10 and 15 minutes after the end of the seizure. The time from the end of succinylcholine administration until return of spontaneous eye opening and obeying verbal commands was recorded. If patient complained of nausea and vomiting inj. Ondansetron 4 mg IV was given. Agitation score was evaluated when the patients were completely awake after ECT, using an emergence agitation score in which 1=sleeping, 2=awake and calm, 3= irritable and crying, 4=inconsolable crying, 5= severe restlessness and disorientation[2]. Incidence and severity of post ECT myalgia was recorded after 6 hrs and 12 hrs. Mild = localized to one group of muscle, moderate= generalized aches and major = interferes with normal activity and mobilization.

The parameters of sessions where propofol was used as induction agent were compiled under the heading of Group P. Similarly findings were compiled as group K and Group KD where Ketofol and ketofol – Dexmedetomidine was used as induction agents, respectively.

Flow chart



Data analysis: Data collection for the study was performed using Microsoft Excel, and subsequent data analysis was carried out using SPSS software version 16. Quantitative data & qualitative data were reported as means ± standard deviations (SD) and as frequencies respectively. The ANOVA test was employed to compare continuous variables between the three groups; p-value of < 0.05 was considered statistically significant.

Ethics: Prior to conducting the study, approval was obtained from the Institutional Ethics Committee. Informed consent was obtained from each patient& their relatives after providing them with comprehensive information about the study, ensuring that they were fully aware of its purpose and procedures. Patients& relatives were assured that their personal information and medical reports would be kept strictly confidential throughout the study.

RESULTS

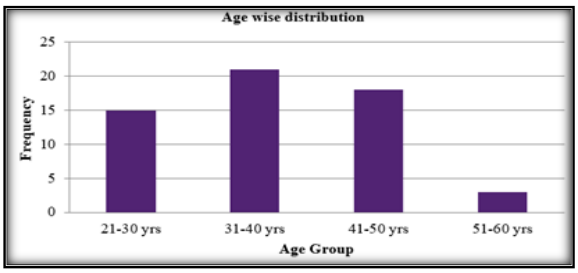


Figure 1: Distribution of patients according to age

The study sample comprised 30 participants with a mean age of 37.47 ± 8.66 years ranging from 22 to 55 years.

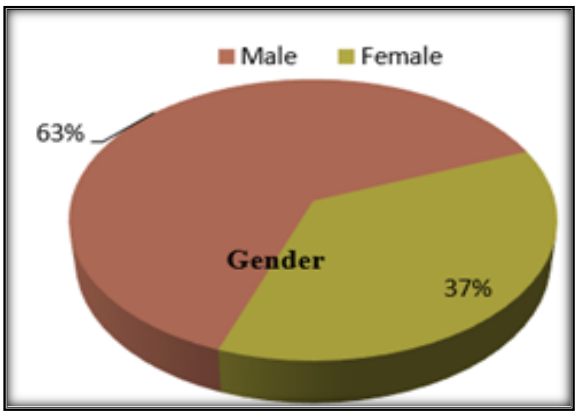


Figure 2: Gender wise distribution of study sample

In our study male to female distribution was 63:32 percentage.

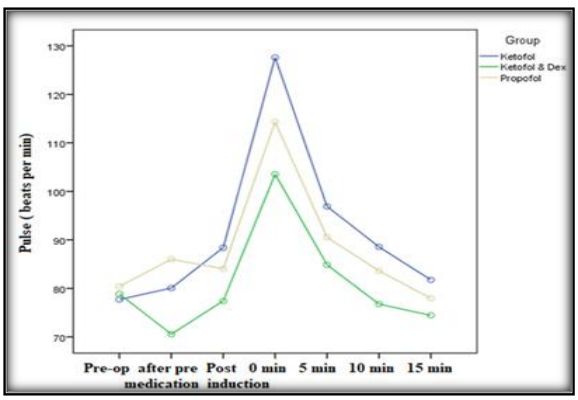


Figure 3: Comparison of heart rate (beats per minute) among study groups

In our study, there was statistically significant difference in heart rate with Group K showing higher value compared to group P and group KD at each time interval after pre medication ($p < 0.05$).

After premedication with Inj. Glycopyrrolate; heart rate increased in all the three groups. Further increase in heart rate was observed after the delivery of shock. Gradually heart rate decreased over period of time but persistently remained on higher side till the end of study period i.e. 15 min after the shock in ketofol group. The percentage increase in heart rate compared to baseline was less in ketofol with dexmedetomidine group than propfol and ketofol group.

We also observed statistically significant difference in mean arterial pressure post induction and at subsequent time interval where group K showed higher value than group P and group KD ($p < 0.05$).

After administration of study drugs there was increase in MAP in Ketofol group whereas Group KD and Group P showed decrease in MAPD. The difference in study group was statistically significant ($P < 0.05$). After delivery of shock there was statistically significant increase in MAP in all the three groups where group K showed maximum increase and group KD showed least values of MAP; the difference being statistically highly significant ($p = 0.001$). MAP values returned to baseline within 5 min. after shock in group KD whereas it took 10 min to normalize MAP in group P and 15 min. in group K.

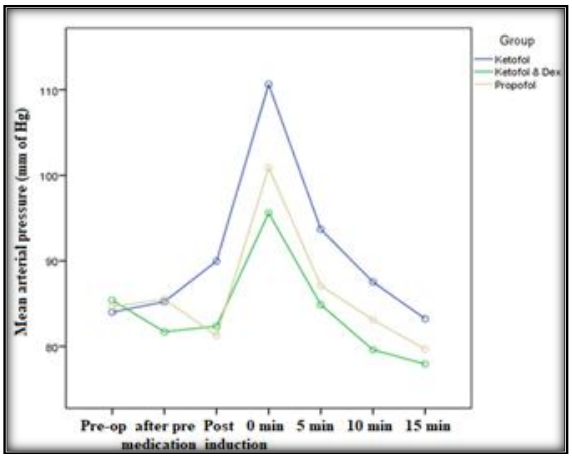


Figure 4: Comparison of mean arterial pressure (mm of Hg) among study groups

Table 1: Comparison of seizure duration and recovery parameters in the three study groups

Parameters	Propofol	Ketofol	Ketofol + Dex	P-Value
	Mean±SD	Mean±SD	Mean±SD	
Seizure Duration (in Seconds)	26.07 ± 7.386	31.87 ± 5.888	42.73 ± 5.948	0.000 HS
Spontaneous Eye Opening time (in minutes)	6.37 ± 2.092	4.63 ± 1.884	9.87 ± 2.688	0.000 HS
Time to Obey Verbal Commands (in minutes)	7.00 ± 2.117	5.10 ± 2.006	11.70 ± 3.075	0.000 HS
Mean Agitation Score	0.70 ± 0..596	2.23± 0.774	0.33 ± 0..479	0.000 HS

HS = highly significant

These results provide insights into the differences in seizure duration and various recovery parameters among the study groups. Ketofol & Dex showed significantly longest mean seizure duration, followed by Ketofol, and then Propofol (p=0.000).Ketofol showed significantly shortest mean time to spontaneous eye opening and time to obey verbal commands, followed by Propofol, and then Ketofol & Dex (p=0.000).Ketofol with dex showed a significantly lower mean agitation score compared to Propofol and Ketofol(p=0.000).

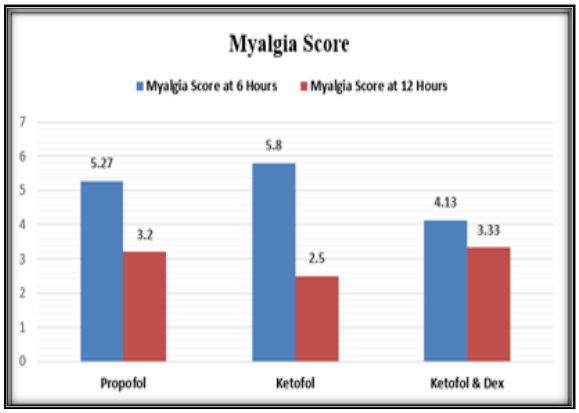


Figure 5: Comparison of myalgia score among study groups

There was statistically highly significant difference in myalgia scores among the different drug treatment groups at 6 hours after ECT with Group KD showing lowest scores ($p = 0.000$). At 12 hours after ECT; all the study groups were comparable with respect to myalgia ($p = 0.198$).

DISCUSSION

In this study, we investigated the efficacy and safety of different inducing agents in electroconvulsive therapy (ECT) by comparing propofol, ketofol, and ketofol with dexmedetomidine.

When we went through the literature; we came across the variability in age distribution and gender ratios across different studies investigating ECT. Such differences are important to consider in clinical practice, as certain age groups or gender distributions may require tailored approaches or considerations during ECT administration. For instance, younger patients might need special attention due to potential differences in seizure threshold or response to anaesthesia, while gender-related factors could influence medication dosages or side effect profiles. To avoid influence of age or gender or any other comorbid conditions of the patient on the outcome of

the study; we decided to study all the three drugs in each & every patient.

Our study sample consisted of 30 participants with a mean age of 37.47 years (SD = 9.66 years), ranging from 22 to 55 years. Among these participants, 19 (63.3%) were male and 11 (36.7%) were female. The largest proportion of participants (46.7%) fell within the 31-40 years age group. Comparing our findings with other studies, Kumar et al., Shams et al. and Gaddam et al. also reported similar mean ages in their studies. In our study male to female distribution was 63:32 percentage.

The data of haemodynamic parameters from our study, along with findings from other studies investigating different drugs in electroconvulsive therapy (ECT), provide valuable insights into the cardiovascular effects of these medications during ECT sessions.

The analysis demonstrated a significant impact of the grouping variable on pulse measurements, with Ketofol showing notably higher pulse rates compared to Ketofol with Dexmedetomidine across various stages, including post-induction. This finding aligns with previous studies.

Comparing our findings with those of other studies, Kayhan et al,[9] observed that ketofol resulted in higher heart rates compared to propofol at T0 (P = 0.03). Shams et al,[10] reported a significant decrease in heart rate in the ketofol- dexmedetomidine group compared to the ketofol group at various time points post-induction (P < 0.01). Dilip et al,[8] found lower heart rates in the propofol group compared to the ketofol group at 1 minute and 5 minutes post-seizure (P = 0.017 and P = 0.032, respectively). Additionally, Yeter et al,[11] noted insignificant changes in heart rate for the ketofol group during ECT, while significant changes were observed in the ketamine-dexmedetomidine group, particularly from baseline to ECT (P = 0.006). Jakhar et al. reported significant increases in heart rate in the ketofol group compared to the propofol group at various time points post-induction (p< 0.05).[12]

Ketofol consistently led to higher heart rate compared to propofol in several studies, while the addition of dexmedetomidine in the ketofol combination results in decreased heart rates compared to ketofol alone.

In our study we observed statistically significant difference in mean arterial pressure post induction and at subsequent time interval where group K showed higher value than group P and group KD.

Comparing our results with those from other studies, Kayhan et al,[9] observed significantly higher MAP values in the ketofol group compared to the propofol group at all time points (P=0.001). Gaddam et al,[5] reported insignificant increases in MAP in the Ketofol group, contrasting with significant rises in MAP in the Thiopentone group and decreases in the Propofol group after induction and shock delivery. Shams et al. [107] observed a significant decrease in MAP in the ketofol- dexmedetomidine group compared to the ketofol group at various time points. Yeter et al,[11] found significant changes in MAP over time in both the Ketofol and Ketamine Dexmedetomidine groups, with differences observed between baseline, ECT 0, ECT 5, and discharge. Jakhar et al,[12] also reported significant differences in MAP between propofol and ketofol groups at various time points post-induction.

Overall, our findings align with previous research indicating varying effects of propofol, ketofol, and ketofol with dexmedetomidine on mean arterial pressure during and after ECT. Ketofol consistently showed higher MAP values compared to propofol in multiple studies, while ketofol- dexmedetomidine results in decreased MAP compared to ketofol alone.

During ECT convulsions lasting between 30 to 60 seconds are considered to be adequate to produce desired therapeutic effect. In our study, we found that the mean seizure duration was significantly longer in the Ketofol with Dexmedetomidine group (42.73 ± 5.948 seconds) compared to the Propofol (26.07 ± 7.386 seconds) and Ketofol (31.87 ± 5.88 seconds) groups, with a P value < 0.05, indicating statistical significance. This suggests that the addition of Dexmedetomidine to Ketofol may prolong seizure duration significantly compared to the other two drugs.

Comparing these results with previous studies, Dilip et al,[8] reported that Ketofol had a longer motor seizure duration (28.55 ± 6.54 seconds) compared to Propofol (22.22 ± 7.94 seconds), which was statistically significant with a P value of 0.002. This aligns with our findings regarding the efficacy of Ketofol in prolonging seizure duration. Yalcin et al,[6] also demonstrated a longer motor seizure duration in the Ketofol group (34 ± 5.8 seconds) compared to Propofol (29.3 ± 5.1 seconds), although both durations were within the therapeutic range. Shams et al,[10] reported a longer motor seizure duration in the Ketofol group (38.9 ± 4.9 seconds) compared to Propofol (35.8 ± 6.6 seconds) with a P value < 0.01, indicating statistical significance. Additionally, Jakhar PM et al,[12] demonstrated a significant difference in seizure durations between Propofol (17.47 seconds) and Ketofol (25.43 seconds) groups with a P value of 0.024. Kumar singh et al,[14] also observed longer seizure duration for Ketofol compared to Propofol (38.93 ± 15.26 vs. 18.10 ± 5.17 seconds; p<0.001).

On the other hand, studies by Kayhan GE et al,[9] and Gaddam et al,[5] did not find a statistically significant difference in motor seizure duration between Ketofol and Propofol groups. Kayhan GE et al. reported durations of 29±17 seconds for Ketofol and 28±13 seconds for Propofol, both deemed adequate for therapeutic efficacy. Similarly, Gaddam et al. found no significant difference in seizure durations between Ketofol (25.88 ± 12.25 seconds) and Propofol (24.85 ± 10.72 seconds) groups.

Overall, the comparison highlights that Ketofol, especially when combined with Dexmedetomidine, tends to prolong seizure duration compared to Propofol in several studies, although the results vary slightly across different investigations.

In our study, we observed a statistically significant difference in eye opening durations among the drug groups. The Propofol group had a mean duration of 6.37 ± 2.092 minutes, the Ketofol group had a mean duration of 4.63 ± 1.884 minutes, and the Ketofol with Dexmedetomidine group had a mean duration of 9.87 ± 2.688 minutes, with a P value < 0.05. This indicates that the addition of Dexmedetomidine to Ketofol significantly prolonged the time to eye opening compared to Propofol and Ketofol alone.

Comparing these results with findings from other studies, Yeter T et al,[11] demonstrated a significant difference in eye opening durations between Ketofol and Ketamine with Dexmedetomidine groups (P < 0.001), with Ketamine with Dexmedetomidine resulting in longer eye opening times.

Dilip et al,[8] Kumar R et al,[13] and Gaddam et al,[5] did not find a statistically significant difference in eye opening durations between Ketofol and Propofol groups (P = 0.311, P = 0.7947, P = 0.431 respectively). Shams et al,[10] and Jakhar PM et al,[12] also reported no significant differences in eye opening durations between Ketofol and Propofol groups (P = 0.174 and P = 0.60, respectively).

In our study, we observed a significant difference in the time to obey verbal command among the three drug groups. Specifically, the mean time to obey verbal command was 7±2.117 minutes in the Propofol group, 5.10±2.006 minutes in the Ketofol group, and 11.7±3.075 minutes in the Ketofol with Dexmedetomidine group, with a P value < 0.05, indicating statistical significance.

Comparing these results with previous studies, Dilip et al,[8] Kumar R et al,[13] Gaddam et al,[5] Shams et al,[10] Kayhan GE et al,[9] Jakhar PM et al,[12] and Yeter T et al,[11] reported varying durations for time to obey verbal command across different drug groups, with some studies showing statistical significance and others not.

Dilip et al,[8] found no significant difference in time to obey verbal command between Ketofol and Propofol groups (P value = 0.768). Kumar R et al,[13] also did not find a statistically significant difference between Ketofol and Propofol groups (P value = 0.0516). Gaddam et al,[5] reported similar findings with no significant difference between Ketofol and Propofol groups (P value = 0.265), as did Shams et al,[10] (P value = 0.260). However, Kayhan GE et al,[9] found a significant difference in time to obey verbal command between Ketofol and Propofol groups (P value = 0.006), indicating superiority ofKetofol. Jakhar PM et al,[12] did not find a significant difference between the two groups (P value = 0.740). Yeter T et al,[11] on the other hand, reported a significant difference between Ketofol (ketamine - propofol) and ketodex (ketamine-dexmedetomidine). Ketamine – dexmedetomidine (p<0.001 and p = 0.003) shows longer duration to obey command.

Overall, the comparison highlights that the addition of ketamine-Dexmedetomidine tends to prolong the time to spontaneous eye opening and time to obey verbal command significantly compared to both Ketofol and Propofol alone, as observed in our study and supported by several studies,[11] although the results vary across different investigations.

Most common adverse effects observed during ECT are post ECT agitation & myalgia.In our study, we observed significant differences in agitation scores among the three drug groups. The mean agitation score was 0.70±0.596 in the Propofol group, 2.23±0.774 in the Ketofol group, and 0.33±0.479 in the Ketofol with Dexmedetomidine group. Ketofol with dexmed (p=0.067) showed a significantly lower mean agitation score compared to Propofol (p=0.000) and Ketofol (p=0.000).

Comparing these results with previous studies, Kumar R et al,[13] reported mean agitation scores of 1.27 ± 0.577 for Propofol, 1.04 ± 0.203 for Ketofol, and 0.0125 for Ketofol with Dexmedetomidine. Shams et al,[10] observed a significantly lower proportion of patients with agitation score > 2 in the Ketofol with Dexmedetomidine group compared to the Ketofol group (1.4% vs. 8.6%, p=0.014). In study by Li X et al,[15] Dexmedetomidine group had lower incidence of post-ECT agitation.The comparison reveals that Ketofol, especially when combined with Dexmedetomidine, may lead to decreased agitation scores compared to Propofol &Ketofol. Our results are similar to abovementioned studies.

There was statistically highly significant difference in myalgia scores among the different drug treatment groups at 6 hours after ECT with Group KD showing lowest scores (p = 0.000). At 12 hours after ECT; all the study groups were comparable with respect to myalgia.

Sriramka et al. also reported that myalgia and fasciculations were less frequent in the Dexmedetomidine group compared to the Normal saline group (P < 0.001).[16]

Overall, the choice of anesthesia agent should consider not only its haemodynamic and sedative properties but also its impact on agitation levels & myalgia. Each drug combination may have varying effects on agitation & myalgia, and clinical judgment based on individual patient factors should guide drug selection to optimize patient comfort and procedural outcomes.

CONCLUSION

The study investigated three different induction agents used in Electroconvulsive Therapy (ECT): Propofol, Ketofol and combination of Ketofol + Dexmedetomidine. Ketofol with dexmedetomidine combination appears to be superior in terms of better hemodynamic stability, increased seizure duration and less incidence of adverse effects although with slightly delayed recovery compared to ketofol and propofol. Therefore Ketofol with dexmedetomidine combination can be used as an effective & safe induction agent during ECT.

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