ETOMIDATE VERSUS COMBINATION OF ETOMIDATE WITH DEXMEDETOMIDINE FOR MOTOR SEIZURE DURATION AND ATTENUATION OF HYPERDYNAMIC RESPONSES TO MODIFIED ECT

Dr. ROJIN SEBASTIAN
Dr. V.S. KAPURKAR, PROFESSOR
DEPARTMENT OF ANAESTHESIOLOGY, KRISHNA INSTITUTE OF MEDICAL SCIENCES, KRISHNA VISHWA VIDYAPEETH (Deemed to be University), KARAD – 415 539, MAHARASHTRA,INDIA

KEYWORDS

ETOMIDATE, DEXMEDETOMIDINE, ECT

ABSTRACT

Introduction: Electroconvulsive therapy (ECT) is a crucial treatment for psychiatric disorders, but concerns about adverse effects have led to research on optimizing anesthesia regimens. This study investigates the efficacy of etomidate alone versus a combination of etomidate and dexmedetomidine in reducing motor seizure duration and mitigating hyperdynamic responses during ECT procedures. **Aims:** The study explores the impact of dexmedetomidine premedication on hyperdynamic responses to modified ECT, seizure duration, and recovery time, aiming to determine its effectiveness. **Methodology:** A prospective, randomized clinical trial at Krishna Institute of Medical Sciences in Maharashtra involved 60 adult patients who underwent modified ECT over 22 months. The study included 30 patients in Group A and 30 in Group B, with etomidate alone or dexmedetomidine premedication as induction agents. Patients were assessed for side effects and discharged post-anesthetic. **Results:** The study compares patients' age, sex, body weight, heart rates, and adverse effects of etomidate alone and etomidate plus dexmedetomidine, finding similar percentages but no significant reduction in rescue analgesia need. **Discussion:** The study reveals a balanced demographic in anesthesia protocols, with males dominating ED and females dominating etomidate-only groups. Combining etomidate and dexmedetomidine reduces adverse effects and enhances patient safety. **Conclusion:** The study found that combining dexmedetomidine with etomidate reduces hyperdynamic response to electroconvulsive therapy without affecting anaesthesia recovery, with lesser nausea, vomiting, and shivering.

INTRODUCTION

Electroconvulsive therapy (ECT) is a crucial treatment for psychiatric disorders like major depressive disorder, bipolar disorder, and schizophrenia, where medication is ineffective. It involves triggering controlled seizures using a short electrical pulse, either directed to one or both brain sides.

The autonomic nervous system is activated, initially slowing heart rate due to parasympathetic nerve activity, followed by a stronger activation of the sympathetic nervous system, causing a temporary increase in heart rate and blood pressure, which can pose risks for patients with heart disease, high blood pressure, or cerebrovascular issues. [1,2]

Despite its effectiveness, concerns about potential adverse effects, such as motor seizure duration and hyperdynamic responses, have led to ongoing research on optimizing anesthesia regimens for ECT procedures. Various medications, including α-2 adrenergic agonists, have been used to reduce stress responses.

Etomidate is a popular anesthetic for ECT due to its favorable hemodynamic profile and rapid action, while dexmedetomidine is known for its sedative and analgesic properties. Cardiovascular reactions to ECT include slowing heart rate, a stronger sympathetic response, rapid heart rate, high blood pressure, and heart muscle issues. [3,4]

Alpha-2 agonists and beta-blockers, particularly for heart or brain conditions, help reduce excessive heart rate and blood pressure response, with clonidine showing promise. [5]

This study investigates the efficacy of etomidate alone versus a combination of etomidate and dexmedetomidine in reducing motor seizure duration and mitigating hyperdynamic responses during ECT procedures. The aim is to improve anesthesia protocols for ECT, enhancing patient safety and treatment efficacy.

This study analyzes clinical outcomes of various anesthesia regimens to guide clinicians towards evidence-based practices in electroconvulsive therapy administration, aiming to optimize patient care and improve treatment outcomes.

AIM & OBJECTIVES

This study investigates if premedication with dexmedetomidine before injection etomidate can reduce hyperdynamic responses to modified ECT and if motor seizure duration remains unchanged during ECT.

The study aims to assess the effects of premedication with dexmedetomidine on hyperdynamic responses to modified ECT, as well as its impact on seizure duration and recovery time.

MATERIALS& METHODS

The study, conducted at Krishna Institute of Medical Sciences' Department of Anesthesiology in Karad, Maharashtra, is a prospective, randomized clinical trial using computer-generated random numbers.

The study was conducted at Krishna Institute of Medical Sciences, KIMSDU, for 22 months, with patients included after written informed consent.

INCLUSION CRITERIA

Patients scheduled for ECT, aged 18-60, with ASA – I & II, no history of beta-receptor blocker drugs or narcotic usage.

EXCLUSION CRITERIA

The exclusion criteria include history of drug allergies, physical status as ASA III, IV, V, baseline bradycardia, cardiovascular diseases, cerebrovascular disorders, hypertension, previous fractures, glaucoma, arterial aneurysm, seizures, and pregnancy.

The study selected a sample size of 30 in Group A and 30 in Group B due to a difference in mean motor seizure duration between the study and control group.

The study involved 60 adult patients who underwent modified ECT over 22 months. They were divided into two groups: Group A, consisting of 30 adults, using etomidate alone as induction agent, and Group B, consisting of 30 adults, using dexmedetomidine premedication followed by etomidate as induction agent.

The procedure involved a detailed pre-anesthetic checkup, including a general physical examination and systemic examination. Patients were informed about the anesthesia technique and given written informed consent. Patients were kept NPO for 6 hours and continued their routine psychiatric medications the night before surgery. The procedure involved standard monitors, including heart rate, ECG, NIBP, and oxygen saturation. Patients were randomly assigned to either group A or B. The procedure involved etomidate 0.2 mg/kg and 0.5 mg/kg succinylcholine induction, with the latter given alone or dexmedetomidine 0.3mcg/kg pre-medication. The procedure involved ECT shock current, which produced seizures. Patients were assessed for side effects and discharged from the post-anesthetic care unit. If intra-procedure SBP, DBP, or MAP was above 160 mm hg, Propofol injection was used as a rescue drug, and Atropine 0.6mg was given if the heart rate was below 50 bpm.

OBSERVATION & RESULTS

Table 1: Distribution of study subjects as per age

Age group	Group				
	ED		E		
	Count	Percent	Count	Percent	Chi square (p value)
<30 years	8	26.7%	6	20.0%	1.73 (0.629)
31-40 years	6	20.0%	9	30.0%	
41-50	9	30.0%	6	20.0%	

years					
51-60 years	7	23.3%	9	30.0%	

The data compares patients' age distribution in two groups: one receiving a combination of etomidate and dexmedetomidine and the other only etomidate. Both groups have similar percentages in each age category, indicating a similar age distribution.

Table 2: Distribution of gender of subjects within the group

Gender	Group				
	ED		E		
	Count	Percent	Count	Percent	Chi square (p value)
F	7	23.3%	5	16.7%	0.417 (0.519)
M	23	76.7%	25	83.3%	

The data shows a sex distribution of patients in two groups: etomidate-and-dexmedetomidine (ED) and etomidate-only (E). Both groups have a similar proportion of males and females, with no statistically significant difference, as indicated by the Chi-square test.

Table 3: Table showing mean comparison of body weight of the subjects between the group

Variable	Group	N	Mean	Std. Deviation	t test	p value
Body weight	E	30	57.500	6.8064	-1.024	.310
	ED	30	59.233	6.2900		

The data presents a comparison of body weight between two groups: one receiving only etomidate (E) and the other receiving a combination of etomidate and dexmedetomidine (ED). The etomidate group has a mean body weight of 57.500 kg with a standard deviation of 6.8064 kg, while the ED group has a mean body weight of 59.233 kg with a standard deviation of 6.2900 kg. The t-test results in a p-value of 0.310, indicating that there is no statistically significant difference in body weight between the two groups. This suggests that both groups have similar body weights on average.

Table 4: Table showing mean comparison of heart rate(bpm) at different time points between the group

Time interval		N	Mean	Std. Deviation	t test	p value
0 min	E	30	78.933	12.2134	.538	.592
	ED	30	77.433	9.1564		
2 min	E	30	92.533	9.4677	1.846	.070
	ED	30	87.967	9.6900		
4 min	E	30	82.400	13.4359	2.779	.007

	ED	30	74.167	9.0975		
6 min	**E**	30	84.667	7.0041	.369	.713
	ED	30	84.000	6.9778		
8 min	**E**	30	84.567	8.4228	2.215	.031
	ED	30	79.633	8.8258		
10 min	**E**	30	92.200	9.3713	2.071	.043
	ED	30	87.133	9.5763		
15 min	**E**	30	82.533	12.5581	2.916	.005
	ED	30	74.267	9.1310		
20 min	**E**	30	84.400	6.8259	.142	.887
	ED	30	84.133	7.6822		
25 min	**E**	30	83.833	6.5236	2.394	.020
	ED	30	79.300	8.0608		
30 min	**E**	30	80.633	12.7860	-.138	.891
	ED	30	81.067	11.5458		
45 min	**E**	30	79.033	12.6777	-.410	.684
	ED	30	80.333	11.8942		
60 min	**E**	30	77.733	15.9177	-.018	.986
	ED	30	77.800	12.1837		

The study compares heart rates between two groups: one given etomidate alone and the other given a combination of etomidate and dexmedetomidine. Results show significant differences in heart rates at specific intervals, with the ED group showing consistently lower rates compared to the E only group. The addition of dexmedetomidine appears to result in lower heart rates.

Table 5: Table showing mean Systolic blood pressure(mmHg) at different time points in both the group

Time interval	**Grp**	**N**	**Mean**	**Std. Deviation**	**p VALUE**
0 min SBP	**E**	30	118.13	7.291	0.51

	ED	30	119.50	8.452	
2 min	**E**	30	117.20	14.072	<0.01
	ED	30	90.73	15.161	
4 min	**E**	30	122.23	26.930	<0.01
	ED	30	95.93	22.228	
6 min	**E**	30	114.50	15.822	0.81
	ED	30	113.57	14.058	
8 min	**E**	30	115.03	16.042	0.96
	ED	30	114.87	14.927	
10 min	**E**	30	118.57	15.815	0.24
	ED	30	114.20	12.780	
15 min	**E**	30	117.87	14.002	0.47
	ED	30	115.53	10.608	
20 min	**E**	30	116.63	11.078	0.87
	ED	30	116.13	13.125	
25 min	**E**	30	117.97	10.662	0.52
	ED	30	116.10	11.868	
30 min	**E**	30	117.13	11.301	0.83
	ED	30	116.53	10.969	
45 min	**E**	30	118.50	12.054	0.68
	ED	30	117.23	11.857	
60 min	**E**	30	117.50	11.904	0.78
	ED	30	116.67	11.742	

The table compares systolic blood pressure (SBP) between two groups: one given Etomidate alone and the other a combination of etomidate and dexmedetomidine. Results show a significant difference at the 4-minute mark, with the Etomidate group having a higher mean SBP. Dexmedetomidine's addition reduces SBP only at the 4-minute mark.

Table 6: Table showing mean comparison of Diastolic blood pressure(mmHg) at different time points in both the group

Time interval	Grp	N	Mean	Std. Deviation	Std. Error Mean	t test	p value
0 min DBP	**E**	30	71.40	6.387	1.166	0.84	.40
	ED	29	73.28	10.299	1.912		
2 min	**E**	30	64.50	4.200	.767	7.01	<0.001
	ED	30	54.93	5.807	1.060		
4 min	**E**	30	73.90	12.291	2.244	4.22	<0.001
	ED	30	61.90	9.561	1.746		
6 min	**E**	30	68.77	9.153	1.671	0.16	.87
	ED	30	69.20	11.639	2.125		
8 min	**E**	30	70.53	8.920	1.629	0.64	.52
	ED	30	68.63	13.443	2.454		
10 min	**E**	30	69.00	9.052	1.653	0.88	.38
	ED	30	66.43	13.146	2.400		
15 min	**E**	30	69.00	7.095	1.295	0.79	0.43
	ED	30	66.87	12.870	2.350		
20 min	**E**	30	68.43	7.001	1.278	0.44	.66
	ED	30	69.47	10.789	1.970		
25 min	**E**	30	68.80	6.945	1.268	0.13	.89
	ED	30	69.13	11.236	2.051		
30 min	**E**	30	68.70	6.959	1.270	0.09	.92
	ED	30	68.50	9.982	1.822		
45 min	**E**	30	68.30	5.528	1.009	0.78	.43
	ED	30	69.93	10.048	1.834		
60 min	**E**	30	67.97	5.021	.917	0.27	.78
	ED	30	68.50	9.217	1.683		

The table compares diastolic blood pressure (DBP) between etomidate alone and etomidate combined with dexmedetomidine. Significant differences were observed at 2, 4, and 6 minutes, indicating lower DBP in the ED group compared to the E group.

Table 7: Table showing mean comparison of Mean atrial pressure(mmHg) at different time points between the group

Time interval	Group	N	Mean	Std. Deviation	t test	p value
0 min	**E**	30	90.27	4.46	-.598	.552
	ED	30	90.93	4.17		

2 min	**E**	30	89.57	4.60	-.273	.786
	ED	30	89.90	4.86		
4 min	**E**	30	91.77	6.055	-.45	.65
	ED	30	91.13	4.710		
6 min	**E**	30	79.00	12.841	-.29	.77
	ED	30	77.97	14.459		
8 min	**E**	30	90.10	4.41	.836	.406
	ED	30	88.87	6.77		
10 min	**E**	30	90.17	5.26	.424	.673
	ED	30	89.53	6.27		
15 min	**E**	30	81.67	11.987	-.26	.79
	ED	30	80.90	10.469		
20 min	**E**	30	90.37	4.93	.077	.939
	ED	30	90.27	5.13		
25 min	**E**	30	90.43	4.45	.347	.730
	ED	30	89.83	8.37		
30 min	**E**	30	77.57	12.336	0.81	.41
	ED	30	80.33	13.790		
45 min	**E**	30	80.83	10.573	0.78	.43
	ED	30	83.20	12.628		
60 min	**E**	30	76.60	10.460	0.78	.434
	ED	30	78.97	12.745		

The study compares mean arterial pressure (MAP) between patients receiving etomidate alone and those receiving a combination of etomidate and dexmedetomidine, finding no significant differences across most time points, indicating comparable values.

Table 8: Table showing mean comparison of SPO2 at different time points between the group

Time Interval	Group	N	Mean	Std. Deviation	t test	p value
0 min	**E**	30	98.37	1.38	-1.785	.079
	ED	30	98.90	0.88		
2 min	**E**	30	98.53	1.14	1.345	0.184

	ED	30	98.13	1.17		
4 min	**E**	30	98.43	1.07	-0.489	0.627
	ED	30	98.57	1.04		
6 min	**E**	30	98.93	1.08	1.816	0.075
	ED	30	98.40	1.19		
8 min	**E**	30	98.47	1.07	-0.336	0.738
	ED	30	98.57	1.22		
10 min	**E**	30	98.30	1.12	0.117	0.907
	ED	30	98.27	1.08		
15 min	**E**	30	98.53	1.14	1.111	0.271
	ED	30	98.20	1.19		
20 min	**E**	30	98.47	1.11	0.122	0.903
	ED	30	98.43	1.01		
25 min	**E**	30	98.87	1.07	1.266	0.211
	ED	30	98.50	1.17		
30 min	**E**	30	98.33	1.09	-0.445	0.658
	ED	30	98.47	1.22		
45 min	**E**	30	98.70	1.06	0.485	0.629
	ED	30	98.57	1.07		
60 min	**E**	30	98.37	1.16	-0.979	0.331
	ED	30	98.67	1.21		

The study compared SPO2 levels between patients receiving etomidate alone and those with a combination of etomidate and dexmedetomidine, finding no significant differences, indicating consistent SPO2 levels throughout the procedure.

Table 9: Table showing mean comparison of motor seizure duration (sec) of the subjects between the group

Variable	**Group**	**N**	**Mean**	**Std. Deviation**	**t test**	**p value**
motor seizure duration(sec)	**E**	30	28.96	8.07	4.352	< 0.001
	ED	30	42.10	14.42		

The study found significant differences in motor seizure duration between patients receiving etomidate alone and those receiving a combination of etomidate and dexmedetomidine, with the ED group experiencing longer seizure durations.

Table 10: Table showing mean comparison of recovery time duration (sec) of the subjects between the group

Group		N	Mean	Std. Deviation	Std. Error Mean	P value
Recovery time (min)	E	30	15.80	.805	.147	0.11
	ED	30	16.17	.950	.173	

A study comparing etomidate and dexmedetomidine recovery times found that the etomidate group had a mean recovery time of 15.80 minutes, while the ED group had a mean recovery time of 16.17 minutes, with no statistically significant difference.

Table 11: Table showing Rescue analgesia needed among the subjects between the groups

			Group		Total
			E	ED	
Rescue analgesia	no	Count	23	27	50
		% Within grp	76.7%	90.0%	83.3%
	yes	Count	7	3	10
		% Within grp	23.3%	10.0%	16.7%
Total		Count	30	30	60
		% Within grp	100.0%	100.0%	100.0%
Chi-sq. value- 1.92, p value- 0.16, non-significant					

The table shows that despite adding Dexmedetomidine to Etomidate, it does not significantly reduce the need for rescue analgesia compared to Etomidate alone, with a Chi-square value of 1.92 and a p-value of 0.16.

Table 12: Table showing adverse effect among the subjects between the groups

			Group		Total
			E	ED	
Adverse effects	Bradycardia	Count	0	4	4
		% Within group	0.0%	13.3%	6.7%
	Drowsiness	Count	0	1	1

		% Within group	0.0%	3.3%	1.7%
	Hypotension	**Count**	0	3	3
		% Within group	0.0%	10.0%	5.0%
	Nausea	**Count**	3	0	3
		% Within group	10.0%	0.0%	5.0%
	Shivering	**Count**	1	0	1
		% Within group	3.3%	0.0%	1.7%
	Vomiting	**Count**	2	0	2
		% Within group	6.7%	0.0%	3.3%
	No adverse effect	**Count**	24	22	46
		% Within group	80.0%	73.3%	76.7%
Total		**Count**	30	30	60
		% Within group	100.0%	100.0%	100.0%

The table compares adverse effects of etomidate alone and etomidate plus dexmedetomidine. Etomidate alone patients experienced higher nausea, vomiting, and shivering, while ED patients experienced bradycardia and hypotension. Overall, 76.7% and 73.3% of patients reported no adverse effects.

DISCUSSION

The age groups in clinical trials are divided into four categories: under 30 years, 31-40 years, 41-50 years, and 51-60 years. The percentages of patients in each age group are similar, with no significant differences. This aligns with Yuerong Peng's 2021 study, Deepa Sannakki's 2017 research, and Keerty Garg's 2015 study, which all found no significant age difference between treatment groups, confirming age as a balanced demographic factor.

The study reveals a gender distribution in anesthesia (ED) and sedative protocols, with males dominating the ED group and females dominating the etomidate-only group. This gender predominance is consistent with previous studies, indicating a skewed gender distribution in clinical settings. The etomidate group has a mean body weight of 57.500 kg, while the ED group has a mean body weight of 59.233 kg. These differences are not statistically significant, suggesting that body weight is a balanced demographic factor.

Heart rate differences are statistically significant between the two groups at different intervals post-administration. The ED group consistently shows lower heart rates at these intervals compared to the Etomidate-only group. However, at other intervals, the differences are not statistically significant. The study suggests that etomidate tends to be associated with higher heart rates compared to dexmedetomidine or propofol, especially at specific time intervals.

Dexmedetomidine is more effective in maintaining lower heart rates during and after procedural interventions, emphasizing the importance of selecting the appropriate sedative agent based on the desired cardiovascular profile.

The study reveals significant differences in systolic blood pressure (SBP) between the Etomidate group and the ED group at the 4-minute mark, with the etomidate group showing a higher mean SBP of 111.83 mmHg compared to the ED group. However, no significant differences were observed at other intervals (0, 2, 6, 8, 10, 15, 20, 25, 30, 45, and 60 minutes). This observation aligns with findings from previous studies, such as Deepa Sannakki's 2017 study, GK Vishwas's 2023 study, and Fangjun Wang's 2022 study.

The study also found significant differences in diastolic blood pressure (DBP) at 2, 4, and 6 minutes, with the etomidate plus dexmedetomidine group showing lower DBP compared to the Etomidate-only group. This suggests that dexmedetomidine may exert a stabilizing influence on DBP during the early stages of the procedure. The combination of etomidate and dexmedetomidine stabilizes DBP more effectively over extended periods, making it a valuable addition in clinical scenarios requiring tight blood pressure management, particularly during procedures involving significant hemodynamic fluctuations.

The mean arterial pressure (MAP) between the etomidate and ED groups showed no statistically significant differences across most time points measured (0, 2, 4, 6, 8, 10, 15, 20, 25, 30, 45, 60 minutes). This finding aligns with Yuerong Peng's 2021 study, where the MAP values for the etomidate and dexmedetomidine groups were also similar across different time points.

Deepa Sannakki's 2017 study provided additional insights into the hemodynamic effects of dexmedetomidine, showing that its administration led to a decrease in MAP to about 85 mmHg post-infusion, compared to the normal saline group whose MAP remained near baseline. The consistency of findings across various studies underscores the reliability of dexmedetomidine in managing hemodynamic stability, making it a valuable option in clinical settings where maintaining stable blood pressure is crucial.

The study found no significant differences in SpO2 levels between the etomidate and ED groups during the observation period (0 to 60 minutes), consistent with previous research by Yuerong Peng. Both anesthetic regimens maintain stable and comparable oxygen saturation levels, which is crucial for patient safety during anesthesia. The addition of dexmedetomidine to etomidate does not adversely affect oxygen saturation, supporting its use in clinical settings where hemodynamic stability is essential.

The mean seizure duration was significantly longer in the ED group compared to the etomidate group, suggesting that dexmedetomidine may potentiate seizure activity during ECT. This finding aligns with the broader literature on the effects of different anesthetic agents on seizure duration during ECT. While dexmedetomidine alone did not significantly extend seizure duration in Garg's study, its combination with etomidate in the present study resulted in significantly longer seizures. These findings are crucial for optimizing anesthetic protocols in ECT to balance efficacy and safety, particularly in terms of seizure quality and duration.

The study found that the mean recovery time for the etomidate group was 15.80 minutes, while the group receiving etomidate plus dexmedetomidine had a mean recovery time of 16.17 minutes. However, Yuerong Peng's 2021 study reported significantly shorter recovery times in various aspects for the dexmedetomidine group compared to the etomidate group. This suggests that dexmedetomidine may contribute to faster recovery from anesthesia compared to etomidate alone, potentially due to its sedative and analgesic properties and ability to reduce anesthetic requirements.

The study also found that in the Etomidate group, 76.7% of patients did not require rescue analgesia, while 23.3% did. In contrast, in the Etomidate plus Dexmedetomidine group, 90.0% of patients did not require rescue analgesia, and 10.0% did. This suggests that the addition of dexmedetomidine to etomidate does not significantly reduce the requirement for rescue analgesia compared to etomidate alone.

The combination of etomidate and dexmedetomidine demonstrated adverse effects like hypotension and bradycardia, while the incidence of adverse effects like nausea and vomiting were more common in the etomidate-only group. The lower incidence of adverse reactions in the dexmedetomidine group aligns with the current study's findings, reinforcing the notion that dexmedetomidine can significantly mitigate the side effects associated with etomidate.

In summary, the current study and Yuerong Peng's 2021 study underscore the advantages of combining dexmedetomidine with etomidate, which appears to significantly reduce the incidence of adverse effects, enhance patient safety and comfort, and suggest that incorporating dexmedetomidine into anesthetic protocols could be a valuable strategy for improving clinical outcomes by minimizing side effects commonly associated with etomidate.

Conclusion

The study found that combining dexmedetomidine with etomidate effectively reduces the hyperdynamic response to electroconvulsive therapy without affecting anaesthesia recovery. Although motor seizure duration was longer in the combination group, it did not significantly impact therapeutic outcomes. Rescue analgesia requirements did not differ significantly between groups. The combination group had a lesser incidence of nausea, vomiting, and shivering, but more frequent bradycardia and hypotension.

Reference

1. American Psychiatric Association. Task Force on Electroconvulsive Therapy. The Practice of ECT: Recommendations for Treatment, Training and Privileging. Convuls Ther. 1990 Jun;6(2):85-120

2. Husain MM, Rush AJ, Fink M, Knapp R, Petrides G, Rummans T, Biggs MM, O'Connor K, Rasmussen K, Litle M, Zhao W, Bernstein HJ, Smith G, Mueller M, McClintock SM, Bailine SH, Kellner CH. Speed of response and remission in major depressive disorder with acute electroconvulsive therapy (ECT): a Consortium for Research in ECT (CORE) report. J Clin Psychiatry. 2004 Apr;65(4):485-91

3. Kellner CH, Knapp R, Husain MM, Rasmussen K, Sampson S, Cullum M, McClintock SM, Tobias KG, Martino C, Mueller M, Bailine SH, Fink M, Petrides G. Bifrontal, bitemporal and right unilateral electrode placement in ECT: randomised trial. Br J Psychiatry. 2010 Mar;196(3):226-34

4. Fink M. Indications for the use of ECT. Psychopharmacol Bull. 1994;30(3):269-75; discussion 276-80

5. Bjølseth TM, Engedal K, Benth JŠ, Dybedal GS, Gaarden TL, Tanum L. Clinical efficacy of formula-based bifrontal versus right unilateral electroconvulsive therapy (ECT) in the treatment of major depression among elderly patients: a pragmatic, randomized, assessor-blinded, controlled trial. J Affect Disord. 2015 Apr 01;175:8-17

6. Qiu Z, Zhou S, Zhang M, et al. Preventive effect of dexmedetomidine on postictal delirium after electroconvulsive therapy: A randomised controlled study. Eur J Anaesthesiol. 2020;37(1):5-13

7. Sannakki D, Dalvi NP, Sannakki S, Parikh DP, Garg SK, Tendolkar B. Effectiveness of dexmedetomidine as premedication prior to electroconvulsive therapy, a Randomized controlled cross over study. Indian J Psychiatry. 2017 Jul-Sep;59(3):370-374

8. Sharan R, Bala N, Attri JP, Garg K. A comparison of dexmedetomidine with propofol versus esmolol with propofol to attenuate the hemodynamic stress responses after electroconvulsive therapy. Indian J Psychiatry. 2017;59(3):366-369

9. Li X, Tan F, Jian CJ, et al. Effects of small-dose dexmedetomidine on hyperdynamic responses to electroconvulsive therapy. J Chin Med Assoc. 2017;80(8):476-481

10. Aksay SS, Bumb JM, Remennik D, et al. Dexmedetomidine for the management of postictal agitation after electroconvulsive therapy with S-ketamine anesthesia. Neuropsychiatr Dis Treat. 2017;13:1389-1394

11. Li X, Tan F, Cheng N, et al. Dexmedetomidine Combined With Intravenous Anesthetics in Electroconvulsive Therapy: A Meta-analysis and Systematic Review. J ECT. 2017;33(3):152-159

12. Parikh DA, Garg SN, Dalvi NP, Surana PP, Sannakki D, Tendolkar BA. Outcome of four pretreatment regimes on hemodynamics during electroconvulsive therapy: A double-blind randomized controlled crossover trial. Ann Card Anaesth. 2017;20(1):93-99

13. Bagle, A. A., Thatte, W., & Kate, P. A. (2016). Dexmedetomidine in premedication to attenuate the acute hyperdynamic response to ECT: a randomised, double-blind, controlled study. Southern African Journal of Anaesthesia and Analgesia, 22(6), 180–184

14. Moshiri E, Modir H, Bagheri N, Mohammadbeigi A, Jamilian H, Eshrati B. Premedication effect of dexmedetomidine and alfentanil on seizure time, recovery duration, and hemodynamic responses in electroconvulsive therapy. Ann Card Anaesth. 2016;19(2):263-268

15. Aydogan MS, Yücel A, Begec Z, Colak YZ, Durmus M. The hemodynamic effects of dexmedetomidine and esmolol in electroconvulsive therapy: a retrospective comparison. J ECT. 2013;29(4):308-311

16. Mizrak A, Koruk S, Ganidagli S, Bulut M, Oner U. Premedication with dexmedetomidine and midazolam attenuates agitation after electroconvulsive therapy. J Anesth. 2009;23(1):6-10

17. Begec Z, Toprak HI, Demirbilek S, Erdil F, Onal D, Ersoy MO. Dexmedetomidine blunts acute hyperdynamic responses to electroconvulsive therapy without altering seizure duration. Acta Anaesthesiol Scand. 2008;52(2):302-306

18. Fu W, White PF. Dexmedetomidine failed to block the acute hyperdynamic response to electroconvulsive therapy. Anesthesiology. 1999;90(2):422-424