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Effectiveness of Dexmedetomidine 0.6 Mics/Kg in Reducing Hemodynamic Stress Response for Patients Undergoing Modified Electroconvulsive Therapy

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I. INTRODUCTION

Psychiatric illnesses are one of the important health problems due to its dysfunction and burden of diseases in developing countries and related to many functional disorders and economical cost in health[1].

Electroconvulsive therapy (ECT), formerly known as electroshock therapy and often referred to as shock treatment, is a standard psychiatric treatment in which seizures are electrically induced in anaesthetized patients to provide relief from psychiatric illnesses like major depressive disorder, schizophrenia, bipolar disorders resistant to medical management[2].

The initial reaction following application of electric current is a parasympathetic response resulting in bradydysartyhmias and possibly sinus pause. The parasympathetic response is followed by a sympathetic response associated with tachycardia and rise in blood pressure as there is a sharp rise in plasma catecholamine levels. During the sympathetic response, systolic blood pressure may increase by 30-40% and heart rate may increase by 20% (or more). The typical effective seizure should have duration of 20 to 50 seconds[3]. The commonest causes of mortality associated with electroconvulsive therapy are the acute changes in heart rate and blood pressure. As a result of the cardiovascular morbidity associated with the electroconvulsive therapy, a wide variety of drugs have been used in an effort to minimize the acute hemodynamic changes[4]. Studies involving trimethaphan[5], nitroprusside[6], nitroglycerine[7], alfentanil[8], clonidine[9], propranolol[10], esmolol[11], labetalol[12,13,14], urapidil[15], dexmedetomidine [16]and diltiazem[17] have reported varying degrees of success in attenuating the acute hyperdynamic response associated with electroconvulsive therapy.

Alpha-2 (α2) adrenergic agonists attenuate stress induced sympathoadrenal responses to painful stimuli, improve intraoperative hemodynamic stability and reduce anaesthetic requirements during surgery. The advantages of intravenous dexmedetomidine as premedicant in anaesthesia setting include sedation, analgesia, anxiolysis and improved hemodynamic stability.

AIM To assess the effectiveness of dexmedetomidine 0.6 mics/kg in reducing hemodynamic stress response for patients undergoing modified electroconvulsive therapy.

OBJECTIVES

Primary Objective

- To measure the peak heart rate, systolic and diastolic blood pressure changes and peak mean arterial pressure following the modified electroconvulsive therapy (ECT).

Secondary Objective

- To study the effect of dexmedetomidine on seizure duration.
- To study the effect of dexmedetomidine on incidence of agitation and patient satisfaction post ECT.

II. MATERIALS AND METHODS

The study was conducted in pre operative area of OT complex of MGM Medical College Navi Mumbai .Study population included were Patients scheduled for ECT belonging to ASA I& II. Patients enrolled in the study were randomly allocated by computer-generated random numbers to receive either dexmedetomidine or saline for their ECT session. The study groups were divided into two, Group C – Control group (normal saline) - 30 patients Group D- Study group (dexmedetomidine 0.6mcg/kg) - 30 patients.

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Sampling technique and sample size calculation: The Open Epi software used for sample size estimation at 95% confidence interval and 80% power of study with the mean RNFL- mean difference in HR between study group and control group .

$$n = 2\frac{S^2(Z1 + Z2)^2}{(M1 - M2)^2}$$

The standard deviation (SD) for HR at T0 interval for study and control group are 9.16 and 11.284 . The estimated sample size is 30 in each group.

Total sample size is 30 x 2=60. In the present study considering the turnover, 30 subjects were enrolled in each group. M1 Mean test intervention M2 Mean control intervention S1 Standard deviation of M1 S2 Standard deviation of M2 S Pooled SD 1-α Set level of confidence

Inclusion Criteria: ASA physical status Iand II **,**age between 18 and 60 years
both male and female gender patients.
Patient's refusal for study, ASA III and above patients, Pregnant and lactating females, patients with heart rate less than 60bpm**,**patients allergic to study medication, patients with difficult airway**,** obese patients (BMI> 30) were excluded from study.

III. MATERIALS

Multichannel monitor with ECG monitoring, pulse oxymeter and Non invasive blood pressure monitor.Inj.Glycopyrrolate 0.2mg, Inj Dexmedetomidine, Inj.Propofol,Inj.Succinyl choline, Guedel's airway, suction catheter no 14 or 16, O2 source,Ambubag,Face mask,Mouth prop, Resuscitation trolley(endotracheal tubes no.6.5,7.0,7.5,8.0,8.5, 9.0, face mask no 3,4, LMA, working laryngoscope , ambu bag, bougie, stylet) .Each patient is evaluated for medical and surgical illness in the past and previous anesthetic exposure and experience.

Following investigations were carried out for all patients.Complete blood count,Urine routine and microscopy,Random blood Sugar,SerumCreatinine,ECG(patients above 40 yrs).

IV. METHODOLOGY

After obtaining the written informed consent, a total of 60 patients confirming to the inclusion and conclusion criteria were included in the study.

Patients were randomly divided into two groups, study group (Group D) who are administered Inj.Dexmedetomidine and control group (Group C) who are administered 0.9% normal saline.

On the day of electroconvulsive therapy, each patient's overnight fasting is confirmed. On arrival of the patient in the electroconvulsive therapy room, ECG, pulse oxymeter and non invasive blood pressure monitors are attached. Baseline heart rate, systolic, diastolic and mean arterial pressures are recorded using a non invasive blood pressure monitor. An intravenous line is secured using 20G/22G i.v cannula.

Ten minutes prior to the electroconvulsive therapy procedure, all patients received premedication Inj.Glycopyrrolate 0.2mg i.v(there were no significant changes in heart rate immediately after Glycopyrrolate) and infusion of 10ml normal saline 0.9% for control group(group C) or Inj.dexmedetomidinei.v 10ml of 0.6mcg/kg slowly over 10 minutes for study group (group D) as per the following table. Patients were pre oxygenated with 100% oxygen through bag and mask. While infusing the study drug, heart rate (HR), systolic blood pressure(SBP), diastolic blood pressure(DBP), mean blood pressure(MAP) and oxygen saturation were monitored at 0^{th}(baseline), 5^{th} min and 10^{th} min after drug administration.

GROUP	Group C	Group D
	10ml Normal Saline	10ml of Dexmedetomidine(0.6mcg/kg)

Table 1: Shows the groups of patients studied and drug received

At the end of infusion, patients were induced with thiopental sodium (3mg/kg) slowly till the loss of consciousness. To one arm pneumatic tourniquet was applied to assess motor seizures. Muscle relaxation was achieved by suxamethonium (0.5mg/kg) and patients were ventilated with 100% oxygen with facemask and ambu bag. A oral soft bite block was inserted and a bi fronto temporal ECT was administered by the psychiatrist. All patients were given the electrical shock current with pulse of 60 to 80 Hz of 0.75msec duration with total stimulus time of 1.25 to 2.5 sec for each ECT. The duration of motor seizure was noted as the time from the electric stimulus to cessation of tonic clonic seizures in the isolated arm. The effectiveness of ECT current was

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verified by appearance of tonic clonic seizures. And the seizure duration was noted. The before mentioned parameters were again noted immediately after induction of anaesthesia (T0), 1st (T1), 3rd (T3), 5th (T5) and 10th (T10) mins after the electrical stimulus. The oral bite block was changed to guedel's airway after the seizure and ventilation was assisted with face mask and 100% oxygen until the return of spontaneous respiration. The patients were observed for 10 minutes in the ECT room and later monitored in the recovery room. Agitation score and satisfaction scale was assessed 10 mins of electrical stimulus.

	HR	ECG	SpO2	SBP	DBP	MAP	Duration of convulsions
Baseline							
5 min after drug study /control drug administration(5th min AD)							
8 min of after study/control drug administration(8th min AD)							
T0 (after 10 mins of AD & immediately after ECT)							
T1							
T3							
T5							
T10							

Table 2: Parameters recorded as per study protocol

Satisfaction Scale

1. Pleased and calm patient
2. Patient without any complaint
3. Patient has some complaint (like headache, nausea etc..)
4. Patient complained that the treatment was unpleasant

Agitation Score

1. Sleeping
2. Awake and Calm
3. Irritable and Crying
4. Inconsolable Crying
5. Severe restlessness and disorientation

Monitoring: The patients were connected to multiparameter monitor to record HR, non-invasive measurements of SBP, DBP, MAP and continuous ECG monitoring and oxygen saturation (SpO2). After stabilization period of 5 minutes, H.R., S.B.P., D.B.P, MAP and SpO2 were measured as baseline parameter. After the administration of study/control drug the HR, SBP, DBP and MAP were recorded at 5th (5th min AD), 8th mins (8th min AD). After 10mins of drug infusion, patients were induced and ECT was given. Above mentioned parameters were noted immediately after ECT at 0, 1, 3, 5 and 10minutes as T0, T1, T3, T5 and T10 respectively.

The times from the ECT stimulus to the cessation of the clonic tonic motor activity in the "isolated" arm (i.e., motor seizure duration) recorded.

After 10 minutes ECT, agitation score and sedation scale are noted.

Statistical Analysis: The data were entered using Microsoft Excel version 2007 and analyzed using Statistical Packages in Social Sciences Software version 20 (SPSS).

Data were expressed as mean ± standard deviation. Quantitative variables were presented as mean ± SD and an unpaired t-test was used to compare significance between the two groups. Qualitative data were expressed as number (%) and analysed using a chi-square test. A p-value of < 0.05 was taken as significant. Data were analysed using Microsoft Excel.

V. RESUTS AND ANALYSIS

In this study, a total of 60 patients of ASA I were selected as per the inclusion and exclusion criteria and randomly divided into two groups of 30 each. Group D received i.v dexmedetomidine 0.6mcg/kg in a total volume of 10ml over 10 minutes and Group C received 10ml of normal

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saline i.v over 10minutes before electroconvulsive therapy
P value of <0.05 was taken as significant.

Demographic Variables: The mean age of patients in the study group (group D) was 32 ± 11.250 years and that in the control group (group C) was 38 ± 13.22 years. The difference in the age between the two groups was not statistically significant (p-value 0.06).

The mean weight of patients in the study group (group D) was 63.6 ± 10.267 kgs and that in the control group (group C) was 66.33 ± 13.845 kgs. The difference in the weight between the two groups was not statistically significant (p-value =0.39).In our study, both the study group and the control groups have 53.3% females, 46.6% males. The females and males are equally distributed across the groups. Thus, the two groups were comparable with respect to sex distribution.
Demographic data was comparable in both the groups.

Heart Rate beats/min

Time	Study Group Mean ± S.D	Control Group Mean ± S.D	p value by Unpaired t test
Baseline Heart Rate	85.27 ± 15.700	80.97 ± 12.417	0.244
5th min AD Heart Rate	82.40 ± 16.454	84.80 ± 12.882	0.532
8th min AD Heart Rate	81.83 ± 16.603	88.20 ± 12.729	0.101
ECT(T0) Heart Rate	107.90 ± 16.998	130.33 ± 20.200	0.01
T1 Heart Rate	100.90 ± 12.474	120.33 ± 15.250	0.01
T3 Heart Rate	93.27 ± 13.81	111.87 ± 14.39	0.01
T5 Heart Rate	89.27 ± 13.653	104.03 ± 15.506	0.01
T10 Heart Rate	84.97 ± 12.941	95.67 ± 15.363	0.01

Table 3: The intergroup comparison of HR (beats/min) changes between study group and control group.

The Baseline HR in both groups are comparable. The baseline HR in study group (group D) was 85.27 ± 15.700 and that in control group (group C) was 80.97 ± 12.417, which was not statistically significant (p=0.244).

Blood Pressure (mmHg)

	Systolic blood pressure(mmHg)		p Value by	Diastolic blood pressure(mmHg)		p Value by
Time	Study Group	Control Group	Unpaired t test	Study group	Control Group	Unpaired t test
Baseline	116.77 ± 11.708	115.33 ± 10.26	0.616	75.43 ± 10.919	74.17 ± 9.628	0.635
5th min AD	112.57 ± 12.201	112.53 ± 9.677	0.991	72.83 ± 10.557	70.53 ± 8.32	0.353
8th min AD	113.43 ± 10.975	116.07 ± 10.232	0.34	72.7 ± 9.596	74.83 ± 10.393	0.412
ECT(T0)	124.07 ± 8.004	133.83 ± 16.686	0.005	81.47 ± 9.38	84.7 ± 12.488	0.261
T1	123.13 ± 13.728	134 ± 15.84	0.006	77.33 ± 10.236	80.33 ± 11.523	0.291
T3	119.57 ± 13.994	129.63 ± 17.645	0.017	75.4 ± 11.761	74.03 ± 11.202	0.647
T5	117.9 ± 14.337	122.3 ± 13.126	0.22	73.53 ± 11.34	71.83 ± 9.826	0.537
T10	112.67 ± 12.807	117.17 ± 12.595	0.175	69.3 ± 9.531	68.27 ± 9.465	0.675

Table 4: The intergroup comparison of SBP and DBP (mmHg) changes between study group and control group

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The baseline SBP (mmHg) in the group D was 116.77 ± 11.708 as compared to group C (115.33 ± 10.26), which was not statistically significant (p=0.616).

SBP in both the groups were comparable at 5th and 8th min AD. SBP at 5th min AD (112.57 ± 12.201 in group D and 112.53 ± 9.677 in group C) and at 8th min AD (group D was 113.43 ± 10.975 and that in group C was 116.07 ± 10.232), are not statistically significant (p value at 5th min AD =0.991 and pvalue=0.34 at 8th min AD).

The mean systolic BP 10 mins after drug administration, induction of anaesthesia and immediately after ECT (T0) in the group D was 124 ± 8.004 which was lower than that in group C 133.83 ± 16.686. The pvalue was statistically significant among the two groups (p=0.006).

Similarly, mean systolic BP at T1 in group D was 123.13 ± 13.728 which was lower than that in group C (134 ± 15.840). The value was statistically significant among the two groups (p=0.006).

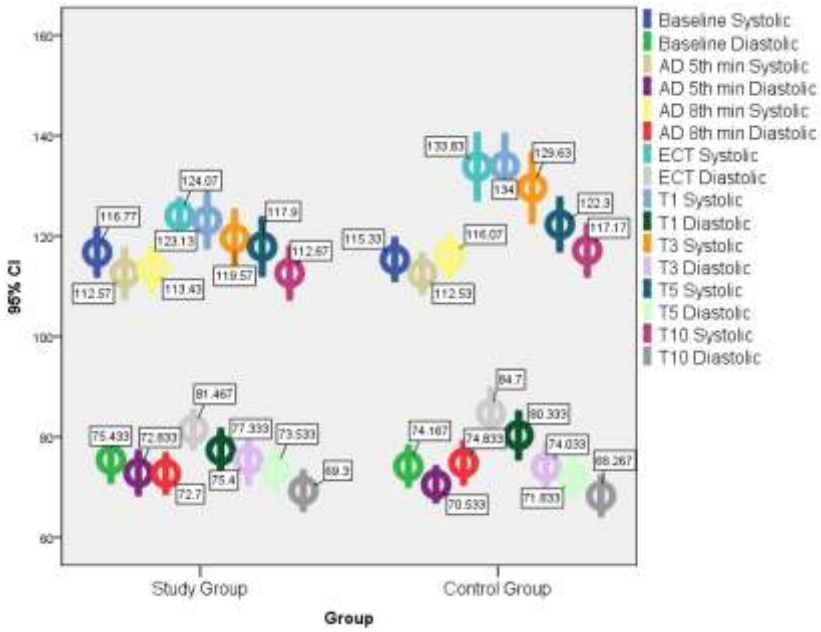
Mean Systolic BP at T3 was 119.57 ± 13.994 in group D as compared to group C was 129.63 ± 17.645, which was also statistically significant among the two groups (p=0.017).

SBP at T5 and T10 in group D was 117.9 ± 14.337, 112.67 ± 12.807 as compared to group C 122.3 ± 13.126, 117.17 ± 12 respectively. These values were not statistically significant (p value= 0.22 at T5 and pvalue= 0.175 at T10) among both the groups.

DBP (mmHg) values are as follows:

Baseline DBP in group D was 75.43 ± 10.919 as compared to the group C 74.17 ± 9.628, which was not statistically significant (p=0.635). 5th min after AD and after 8th min AD in group D was 72.83 ± 10.557, 72.7 ± 9.596 as compared to the group C was 70.53 ± 8.32, 74.83 ± 10.393 respectively. These values were not statistically significant (p=0.353 at 5th min AD, p=0.412 at 8th min AD).

DBP (mmHg)10 mins after drug administration, induction of anaesthesia and immediately after ECT (T0), T1, T3, T5 and T10 in the group D was 81.47 ± 9.38, 77.33 ± 10.236, 75.4 ± 11.761, 73.53 ± 11.34 and 69.3 ± 9.531, as compared to the group C was 84.7 ± 12.488, 80.33 ± 11.523, 74.03 ± 11.202, 71.83 ± 9.826 and 68.27 ± 9.465 respectively. These values were not statistically significant (p=0.261 at T0, p=0.291 at T1, p=0.647 at T3, p=0.537 at T5 and p=0.675 at T10) among the two groups.



Graph 1: Graphical representation of intergroup comparison of Systolic and diastolic blood pressure.

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Mean arterial pressure (mm of Hg)

	Study Group	Control Group	p Value by Unpaired t test
Baseline MAP	89.97 ± 10.407	89.03 ± 8.884	0.71
5th min AD MAP	86.03 ± 10.581	85.2 ± 7.444	0.726
8th min AD MAP	87.23 ± 9.551	89.33 ± 9.838	0.405
ECT(T0) MAP	97.1 ± 7.246	104.07 ± 14.846	0.024
T1 MAP	94.5 ± 9.73	99.73 ± 11.383	0.061
T3 MAP	91 ± 11.507	93.33 ± 10.784	0.421
T5 MAP	88.13 ± 11.307	90.53 ± 9.755	0.382
T10 MAP	84.03 ± 8.992	85.93 ± 8.796	0.411

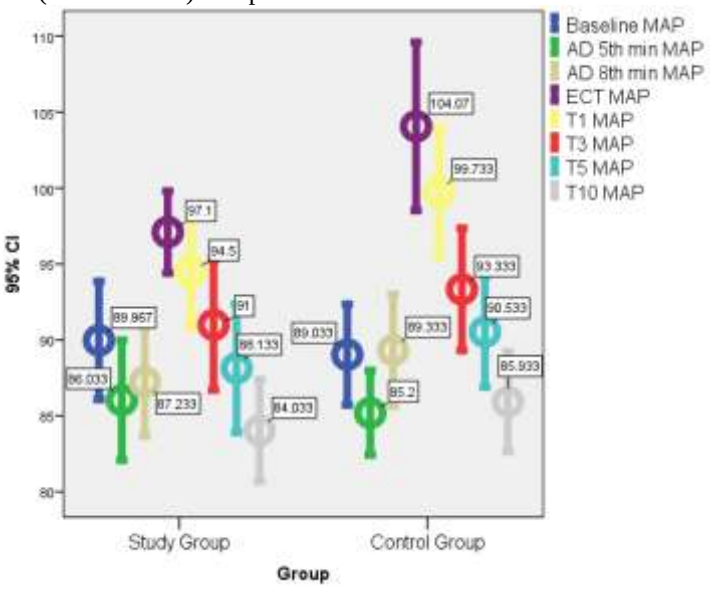
Table 5: The intergroup comparison of MAP (mmHg) changes between the two groups

The baseline MAP (mmHg) in group D was 89.97 ± 10.407 and in group C was 89.03 ± 8.884. The values were comparable and not statistically significant (p=0.71).

MAP in group D at 5th min AD (86.03 ± 10.581) and 8th min AD (87.23 ± 9.551) compared to group C at 5th min AD (85.2 ± 7.444) and at 8th min AD (89.33 ± 9.838). There was no statistical significant difference between the groups (p=0.726 at 5th min AD and p=0.405 at 8th min AD). MAP in both groups before induction were comparable.

There was a decrease in MAP value during T0 in the group D (97.1 ± 7.246) compared to group C (104.07 ± 14.846) which was statistically significant (p=0.024).

Thereafter, MAP values in group D were 94.5 ± 9.73, 91 ± 11.507, 88.13 ± 11.307 and 84.03 ± 8.992 compared to the group C 99.73 ± 11.383, 93.33 ± 10.784, 90.53 ± 9.755, and 85.93 ± 8.796 at T1, T3, T5 and T10 respectively. These values are not statistically significant (p=0.061 at T1, p=0.421 at T3, p=0.382 at T5, p=0.411 at T10) among the two groups.



Graph 2: Graphical representation of intergroup comparison of MAP.

Seizure duration (in seconds)

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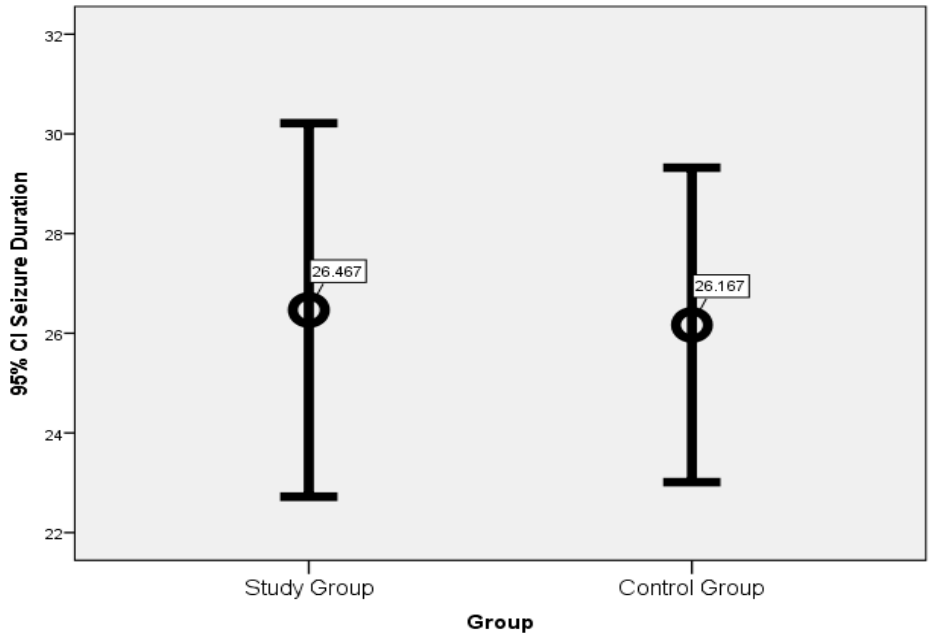
Seizure Duration(seconds)		
Study Group	**Control Group**	**p Value by Unpaired t test**
26.47 ± 10.034	26.17 ± 8.449	0.901

Table 6: Intergroup comparison of seizure duration (seconds)

Seizure duration in group D was 26.47 ± 10.034 seconds as compared to the group C which was 26.17 ± 8.449 seconds. There was no statistically significant difference (p=0.901) in seizure duration among two groups.



Graph 3: Graphical representation of seizure duration (seconds)

Satisfaction Scale

Patient's satisfaction was evaluated, when the patients were awake completely after 10mins of ECT and were assessed using satisfaction scale.

Satisfaction Scale

1. Pleased and calm patient
2. Patient without any complaint
3. Patient has some complaint
4. Patient complained that the treatment was unpleasant

As seen in the above table 8, 11 patients (36.7%) in group D were calm and pleased after ECT as compared to 6 patients (20%) in group C. 19 (63.3%) patients were satisfied without any complaint in the group D as compared to 18 (60%) patients in control group C. In the group D, no patient had any complain after ECT and no patient complained that the treatment was unpleasant. In the group C, 6 patients (20%) had minor complaints like headache and nausea after ECT, but no patient complained that the treatment was unpleasant.

	Group	N	Mean	Std. Deviation	Std. Error Mean	p Value by unpaired t test
SatisfactionScale	**Study Group**	30	1.63	0.490	0.089	0.02
	Control Group	30	2.00	0.643	0.117	

Table 7: Satisfaction Scale

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Group D had a satisfaction score of 1.63 ± 0.490 as compared to 2.00 ± 0.643 in group C. Patient's satisfaction scale was better in group D as compared to the group C. The result was statistically significant (pvalue=0.02) as shown in table 13.

Agitation Score

Patients were evaluated for agitation using emergence agitation score after 10 mins of ECT.

Agitation Score:

1- Sleeping
2- Awake and calm
3- Irritable and crying
4- Inconsolable crying
5- Severe restlessness and disorientation

In group D, 16 patients (53.3%) were sleeping as compared 4 patients (13.3%) in the group C. 13 patients (43.3%) had agitation score of 2 (awake and calm) in the group D compared to 20 patients (66.67%) in group C. Only 1 patient (3.3%) had score of 3 (irritable and crying) in group D as compared to 6 patients (20%) in the group C. No patient in any group had a score of 4 (inconsolable crying) or 5 (severe restlessness and disorientation) as shown in table 14.

	Group	N	Mean	Std. Deviation	Std. Error Mean	P Value by unpaired t test
Agitation Score	Study group	30	1.50	0.572	0.104	0.01
	Control group	30	2.07	0.583	0.106	

Table 8: Agitation Score

Emergence agitation score assessed 10 mins after ECT was 1.50 ± 0.572 in group D which was lower compared to 2.07 ± 0.583 in group C. This difference was statistically significant (pvalue=0.01) as described in table 9 and 10.

VI. DISCUSSION

Almost all ECT procedures are performed under general anaesthesia with muscle paralysis. ECT induces tonic clonic epileptic seizure. Variety of adverse physiologic and physical effects occur due to electric current applied to the brain. Cardiovascular and central nervous system responses are potentially most dangerous

The typical cardiovascular response to ECT consists of generalized autonomic nervous system stimulation, with an initial parasympathetic-induced bra dycardia lasting 10 to 15seconds followed immediately by a more prominent sympathetic response that results in transient tachycardia and hypertension lasting 5 mins or longer.

Systolic blood pressure (SBP) is transiently increased by 30%–40% and heart rate (HR) is increased by 20% or more, resulting in a two to four fold increase in the rate-pressure product (RPP), an index of myocardial oxygen consumption[4]. These hemodynamic responses predispose vulnerable patients to significant cardiovascular complications like cardiac dysrhythmias, myocardial ischaemia and infarction.

. Dexmedetomidine has shown to be promising in various procedures to blunt this catecholamine-induced stress response[39]. Hence, we decided to use dexmedetomidine as premedication in our study.

Aparna Bagle et al[18] conducted a study using Inj Dexmedetomidine 0.5mcg/kg as pre-treatment for ECT and found that the mean agitation score was significantly lesser in the Dexmedetomidine group (1.5 ± 0.5) as compared to the control group (1.93 ± 0.52). The p value=0.0018, which is statistically significant. This is similar to our results.

Xiang et al[38] observed that MAP values at 10min after ECT in both the groups is comparable (p=0.353). This is similar to our study.

In a study conducted by Deepa Sanakki et al[39] observed increase MAP values in control group at 1min, 3min, 5min and 10mins after ECT as compared to Dex group. These values are statistically significant (p<0.05). These results are in contrary to our results. This may be because of use of higher dexmedetomidine concentration (1mcg/kg) as pretreatment, as compared to our study (0.6mcg/kg).

Seizure Duration

In our study, the mean seizure duration in the study group is 26.47± 10.034 compared to 26.14 ± 8.449 in control group. The difference between two groups is not statistically significant (p=0.901).

Although the exact mechanism of the therapeutic action of ECT is unknown, the seizure duration is regarded as an indicator of the efficacy of ECT. Formerly, a seizure duration of >25 seconds has been recommended to ensure clinical adequacy of ECT. Most short acting anaesthetics used in ECT decrease the seizure duration in a dose dependant fashion[32].

In the study conducted by Begec et al[30] the motor seizure duration in the control group (36 ± 15s) which is similar to that in the dexmedetomidine group (33 ±12 s) (p>0.05)[32]. This observation is similar to the finding of our study.

Satisfaction Scale

Satisfaction scale was observed 10mins after ECT. 36.7% patients in the study group were pleased and calm as compared to 20% patients in the control group.

In the study group remaining 63.3% patients were without any complaint compared to 60.0% patients in the control group.

There was no patient in the study group who had any complaint after ECT, whereas 20% patients had complaints like headache and nausea in the control group.

No patient in any group complained that the treatment was unpleasant.

Group D has a satisfaction score of 1.63 ± 0.490 as compared to 2.00 ± 0.643 in group C. Patient's satisfaction is better in group D than in group C and the result being statistically significant (pvalue=0.02) as shown in table 9.

Agitation Score

Agitation score was assessed 10 mins after ECT.

In the study group, 53.3% patients had score of 1 (sleeping) compared to only 13.3% patients in the control group.

In study group, 43.3% patients were awake and calm (score 2) in comparison with 66.7% patients in control group.

Only 1 patient (3.3%) was irritable and crying (score 3) in study group, whereas 6 patients (20.0%) were irritable and crying (score 3) in the control group. No patient in any group had a score of 4 (inconsolable crying) or 5 (severe restlessness and disorientation).

The agitation score in group D was 1.50 ± 0.572 and in group C was 2.07 ± 0.583.Thus the agitation score was lower in group D as compared to group C, which is statistically significant (p=0.01). This suggests that premedication with Inj Dexmedetomidine significantly reduces post-ictal agitation in patients undergoing ECT.

VII. CONCLUSION

The current study was conducted at MGM Medical College Navi Mumbai, to study the effectiveness of pre-medication of dexmedetomidine in attenuating acute hemodynamic stress response to ECT, we can conclude that:-

- Dexmedetomidine at a dose of 0.6mcg/kg intravenously, leads to a significant reduction in HR, SBP and MAP responses to ECT.
- Dexmedetomidine doesn't alter the therapeutic seizure duration.
- Low dose dexmedetomidine effectively reduces the incidence rate of post-ECT agitation.
- Pre-medication with low dose dexmedetomidine intravenously before ECT may be useful for better patient satisfaction post-ECT with no side effects.

Thus, the current study provides new insights into the clinical use of dexmedetomidine in ECT. Premedication with 0.6mcg/kg dexmedetomidine intravenously before the ECT effectively controls the hemodynamic responses to ECT without altering seizure duration. In addition low dose of dexmedetomidine effectively reduces the incidence of post-ECT agitation and better patient satisfaction score with no side effects.

LIMITATIONS OF THE STUDYThis is a small numbered, single hospital based control study, generalization of result is difficult to predict.The comparison of different doses of dexmedetomidine was not studied. EEG seizure duration was not recorded, but only motor seizure duration was recorded

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