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ATTENUATION OF HEMODYNAMIC RESPONSE IN MODIFIED ECT USING DEXMEDETOMIDINE

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ABSTRACT

BACKGROUND: Electroconvulsive therapy is used to treat psychiatric disorders associated with initial parasympathetic response later by a sympathetic response like tachycardia and hypertension which can results in myocardial infarction and cardiac arrest in vulnerable people. The objective of our study to evaluate the efficacy of dexmedetomidine in attenuating the hemodynamic response to modified ECT and its effect on seizure duration, time of recovery and the agitation score.

MATERIALS AND METHODS: This randomised prospective double blinded cross over study was carried out in 50 patients, divided into two groups (the same patient acts as both control and study in separate sittings). Group C received 100ml of plain normal saline intravenously over 10min,and Group D received dexmedetomidine 0.5µg/Kg in 100ml saline 10 minutes prior to induction. Anaesthesia was standardized in both groups and vital parameters, Motor seizure duration, the time taken for the recovery of the patient and agitation score were recorded at 1, 2, 5,10,20,30,60,minutes and 2,4 hours after the ECT shock.

RESULTS: There was statistically significant fall in SBP, DBP, (MAP), heart rate in the dexmedetomidine group (p value=0.0006), (p value=0.004), (p value=0.013) and (p value -0.0001) respectively. The agitation score was lower in group D compared to group C which was statistically significant (p value=0.0001) but no significant difference in saturation (p value >0.05).

CONCLUSION: Dexmedetomidine at a dose of 0.5µg/Kg given before Modified ECT attenuates the hyper dynamic response with a slight delay in time for recovery.

KEYWORDS : Modified ECT, Dexmedetomidine, Attenuation of haemodynamic response

INTRODUCTION

ECT is a standard psychiatric treatment developed in 1938 where a generalized epileptic seizure is purposely induced for the treatment of psychiatric disorders that are resistant to medical management [2 3]. In "Modified ECT " usually short acing anaesthetics are given in addition to muscle relaxant [5]. On application of the electric current there is a parasympathetic response which is followed by a sympathetic response like systolic blood pressure may increase by 30-40% and heart rate may increase by 20% or more [10]. Patient with coexisting disease like hypertension, coronary artery disease may develop complications like disturbance of heart rhythm, left ventricular failure, myocardial infarction and cardiac arrest. Many drugs were tried to attenuate hemodynamic response during ECT [13,14]. Clonidine and dexmedetomidine are centrally acting α-2 adrenergic agonists attenuate stress induced sympatho adrenal responses to painful stimuli, improve intraoperative hemodynamic stability, and reduce anaesthetic requirements[16, 22].Dexmedetomidnine is an extremely elective α-2 receptor agonist having eight times higher affinity than clonidine, different benefits of dexmedetomidine are anxiolytic sedative agent in ICU (16),post operative care unit and analgesic with better haemodynamic control without any respiratory depression [13,14].

MATERIALS AND METHODS

This is a randomised prospective double blinded cross over study. This was conducted in the Psychiatry Department of Mahatma Gandhi Memorial Government Hospital, Tiruchy in patients scheduled for ECT. Formal Ethical Committee approval was obtained. The period of the study was from August 2014- July 2015.

INCLUSION CRITERIA

Age 18-60 years with psychiatric disorders (Schizophrenia, Mania, and Depression) with ASA I-II Physical status scheduled for ECT

EXCLUSION CRITERIA

Serious physical disease like cardiovascular disease, cerebrovascular disease, Intracranial hypertension, Respiratory tract disease, Previous fracture, Glaucoma, Arterial aneurysm, Cerebrovascular malformations, History of seizures, Pregnancy, patients with difficult airway, Obese patients (BMI>30). Patients with endocrinal diseases like hyperthyroidism, hypothyroidism, and Diabetes mellitus.

SPECIFIC TO THE DRUG

Patients with HR less than 60 bpm
SBP less than 100 mm of Hg
History of allergy to study drugs
History of cardiac conduction defects
ASA III –V Physical status

METHODOLOGY:

A total of 50 patients were scheduled for the study. The study is cross-over study in which the same patient acts as both control and study in separate sittings of ECT (total sample was 100). After getting basic investigations results, Pre-anaesthetic evaluation was done .A written informed consent was obtained on the day before the procedure and the study was done in repeated crossover pattern. Same sample of patients received saline in one sitting of ECT and dexmedetomidine in second sittings. In ECT room drugs were prepared and labelled as A and B by a senior anaesthesiologist not involved in the study. Administering drugs and monitoring were done by a separate investigator who did not know about the preparation. In the first sitting of ECT 50% of the sample received drug A and another 50% drug B. In the second sitting the patients who received drug A were given drug B and vice versa. Later the syringes were discarded. All the patients when received saline were allocated as Group C and same patients when received dexmedetomidine as Group D

GROUP C: Saline group
GROUP D: Dexmedetomidine group
Group D received dexmedetomidine 0.5µg/Kg in 100ml saline and Group C

All the patients were pre medicated with tablet ranitidine 150mg and kept in adequate starvation previous night before procedure. Antipsychotic and antidepressants were continued on the day of procedure. On the morning, Inj. Glycopyrrolate 0.2mg was given I'm 30 minutes before the procedure to decrease the secretions and attenuate the parasympathetic response of ECT. Patients were received 100ml of plain normal saline intravenously 10 minutes prior to induction over the period of 10 minutes, Inj. Atropine and Inj. Ephedrine were kept loaded in syringes in case the parasympathetic response is intense resulting in severe bradycardia and hypotension. On arrival of the patient in the ECT room, an 18 gauge cannula was inserted and an infusion of normal saline was started. The patients were connected to multi parameter monitor to record HR, non-invasive measurements of SBP, DBP, MAP, continuous ECG monitoring and oxygen saturation (SpO_2). After stabilization period of 5 minutes, HR, SBP, DBP, MAP and SpO_2 were measured as baseline parameter. Then either the study drug or saline was administered and hemodynamic

parameters were recorded at 0, 5 and 10 minutes. Patient was pre oxygenated for 3minutes via face mask with Bain's circuit. Anaesthesia was induced with propofol 1mg/Kg until the patient loses consciousness followed by 0.5mg/Kg of succinylcholine succinyl choline. The stimulus electrodes were applied on either side of the scalp. An oral soft bite block was placed and ECT shock current was applied. All the patients were given the electrical shock current with a pulse of 60–80Hz of 0.75msec duration with total stimulus time of 1.25–2.5 seconds for each ECT [3,9]. Seizure duration may be affected by ECT stimulus setting of ECT Machine. To control this factor, all patients were given ECT by same Machine and with fixed ECT stimulus settings The effectiveness of ECT current was verified by appearance of tonic - clonic seizures and by the EEG recording in the ECT machine. Controlled or assisted ventilation was continued with 100% oxygen (5 litres /min.) until patient resumed adequate spontaneous breathing. The HR, SBP, DBP and MAP recorded at 1, 2, 5, 10, 20, 30, 60 minutes and 2, 4 hoursrs after the ECT shock. The time from the ECT stimulus to the cessation of the clonic tonic motor activity in the "isolated" foot (i.e., motor seizure duration) recorded. The time taken for the recovery of the patient (from the end of succinylcholine administration until the resumption of spontaneous breathing, eye opening, and obeying commands) were recorded. Agitation score [1,25,26] was evaluated when the patients were completely awake after ETC.

The agitation was evaluated using an emergence agitation score
1 = Sleeping
2 = Awake and calm,
3 = Irritable and crying
4 = Inconsolable crying
5 = Severe restlessness and disorientation [57]

STATISTICALANALYSIS:

was done by SPSS version 21.0 software and Student t test were applied for the interpretation of results. A P value < 0.05 was considered as statistically significant.

OBSERVATION AND RESULTS

This is a cross over study comparing the effects of dexmedetomidine on the hemodynamic parameters during modified ECT for 100 patients. Each patient serves as his /her own control.

DEMOGRAPHIC DATA

Patients between the age group of 20 – 51 years was preferred As this is a crossover study, the mean age of the patients participated in the study was 33.3 ± 8.2 and the mean weight was about 53.2 ± 5.51.Out of the 50 patients participated in the study 30 were male (60%) and 20 were female (40%) The distribution of the ASA status in the study population was ASA I about 48 patients (96%) and ASA II was 2 patients (4%)

Table. 1 Preoperative Status

	HR	*SBP*	*DBP*	*MAP*	*SPO2*
GROUP C	97.7 ± 9.7	118.2 ± 8.2	80.3 ± 4.8	94.6 ± 5.9	98.8 ± 1.1
GROUP C	96.2 ± 11.1	117.7 ± 9.3	81.4 ± 5.1	95.8 ± 6.3	99.1 ± 0.9
t value	0.772	0.303	-1.110	- 1.211	-1.492
P value	0.471	0.762	0.265	0.228	0.138

	HR			**SBP**			**DBP**			**MAP**			**Spo2**		
MIN	**0**	**5**	**10**	**0**	**5**	**10**	**0**	**5**	**10**	**0**	**5**	**10**	**0**	**5**	**10**
GROUP C	92.9 ± 8.2	92.7 ± 5.6	92.2 ± 6.3	121.3 ± 8.1	120.5 ± 6.8	120.3 ± 5.7	79.9 ± 4.3	79.7 ± 4.5	79.7 ± 4.8	93.4 ± 4.6	93.1± 4.9	93.2 ± 5.1	99.1 ± 0.9	98.8 ± 0.6	99.0 ± 0.5
GROUP D	91.3 ± 7.9	86.4 ± 4.5	82.2 ± 5.2	118.6 ± 7.9	118.4 ± 6.0	116.1 ± 6.2	78.7± 4.7	78.2 ± 4.9	76.8 ± 4.5	92.8 ± 4.3	92.2 ± 4.2	90.7 ± 4.8	98.9 ± 0.7	98.7 ± 0.4	98.8 ± 0.8
T VALUE	0.993	6.201	8.656	1.687	1.637	3.526	1.332	1.594	2.901	0.673	0.986	2.524	1.240	0.877	1.499
P VALUE	0.32> 0.05	**0.0001 <0.05**	**0.001< 0.05**	0.094 > 0.05	0.104 > 0.05	**0.0006 < 0.05**	0.185 >0.05	0.114 >0.05	**0.004 < 0.05**	0.503 >0.05	0.324 >0.05	**0.013 < 0.05**	0.217 > 0.05	0.382 > 0.05	0.137 > 0.05

The baseline HR, SBP, DBP, MAP and SPO2 in the patients does not show any significant change in the two sittings. These values were compared since the antipsychotics drugs are parasympathomimetics which may cause a change in these parameters. After recording baseline parameters, either saline or dexmedetomidine were infused and the hemodynamic parameters noted

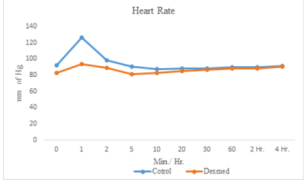
Table 2:Infusion of Saline / Dexmedetomidine Hemodynamic variables

INTRAOPERATIVE HEMODYNAMICS

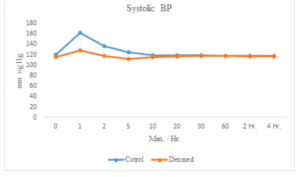
Intraoperative hemodynamic parameters (HR,SBP,DBP,MAP,SPO_2) were noted just before the application of the ECT stimulus and after 1,2 ,5,10,20,30,60 minutes 2 and 4 hrs after the end of seizure. The results observed were compared between the two groups

HEART RATE (HR)

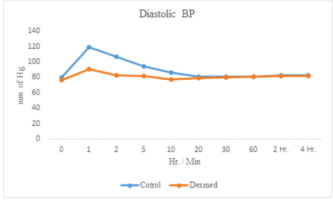
The mean HR increase observed at 0,1, 2, 5 and 10minutes after ECT shock in group C was statistically highly significant compared to mean HR increase in group D (p=0.0001).



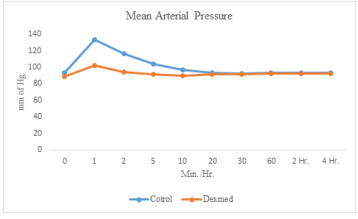
SBP



DBP



MAP



The SPB, DBP and MAP were significantly lower in group D compared to group C after drug administration at 5 and 10 minutes. The increase in SBP, DBP and MAP in group C was statisticallyhighly significant at 0, 1.2, 5 and 10 minutes after ECT shock (p=0.000) compared to groupD.

Table 3. Saturation SPO_2

Time	Group C Mean ± S.D	Group D Mean ± S.D	T value	P value
0 Min.	98.6 ± 0.9	98.5 ± 0.7	0.6202	0.5366
1 Min.	98.8 ± 0.5	9.9 ± 0.3	1.2127	0.2282
2 Min.	98.5 ± 0.4	98.7 ± 0.6	1.9612	0.0527
5 Min.	98.6 ± 0.7	98.5 ± 0.8	0.6652	0.5075
10 Min.	99.6 ± 0.5	99.5 ± 0.4	0.7634	0.3276
20 Min.	99.3 ± 0.3	99.4 ± 0.5	1.8452	0.0891
30 Min.	99.2 ± 0.8	99.1 ± 0.2	0.5367	0.0912

60 Min.	99.1 ± 0.7	99.2 ± 0.8	0.6652	0.5075
2 Hr.	99.5 ± 0.3	99.4 ± 0.2	1.9612	0.0527
4 Hr.	99.5 ± 0.2	99.4 ± 0.3	1.8263	0.0734

There was no significant difference in the saturation between the two groups at any time after the drug administration and the ECT stimulus (p value >0.05) (Table.3).

Table 4. Seizure Duration (Sec)

Groups	Mean ± S.D	Statistical Inference
Group C	36.9 ± 3.5	T value 0.189
Group D	37.8 ± 3.2	P value 0.849

The duration of the seizure is noted since the occurrence to the cessation of the clonic tonic motor activity in the isolated foot. There is no statistically significant difference in the seizure duration between the two groups. (P value = 0.849) (Table 4).

Table 5. Time for Recovery (Min.)

Groups	Mean ± S.D	Statistical Inference
Group C	10.6 ± 2.5	T value 6.210
Group D	35.8 ± 3.2	P value 0.0001

The time taken for the recovery of the patient from the end of succinylcholine administration until the resumption of consciousness and obeying commands were recorded. The time to recovery was greater in the group D compared to group C which is statistically significant (p value=0.0001) (Table 4).

Table 5. Agitation Score

Groups	Mean ± S.D	Statistical Inference
Group C	1.40 ± 0.7	t value 3.011
Group D	1.08 ± 0.2	p value 0.03

The post ictal agitation is scored using an agitation score from 1 to 5.The agitation score is lower in group D compared to group C which is statistically significant. (p-value=0.0001) (Table.5)**.**

DISCUSSION

In this study the effectiveness of dexmedetomidine to blunt the hemodynamic insult and its additional benefit on preventing hemodynamic surge associated with neuro hormonal response of ECT was evaluated [13,14,18]. We found that there is a fall in heart rate at the 5th and the 10th minute and a significant drop in SBP, DBP, MAP at the 10th minute after the drug administration, after the administration of the ECT stimulus the mean rise in HR SBP,DBP,MAP was lower in the dexmedetomidine group when compared with the saline group at 0,1,2,5,10 These results are consistent with the results of the studies by Zakine Begec *et al*. and Ravichandra Dodawad *et al,*in which they studied the effect of 1µg/Kg of dexmedetomidine, 0.6µg/Kg of dexmedetomidine (in 100 patients) to attenuate the hemodynamic response to ECT respectively. They found that HR, MAP in the dexmedetomidine group was lower than that in the control group at 5 and 10 min and also, SBP, DBP at 5 and 8 min.

Also in a study by Tarek Shams *et al*., compared ketofol and ketofol-dexmed mixture in 40 patients scheduled for ECT (Dexmedetomidine 0.5µg/Kg).There was a significant decrease in heart rate ($P < 0.01$) in ketofol-dex group compared to ketofol group 5,10,20, and also decrease in MAP is observed only after 20 minutes (Table.5). There was no difference in oxygen saturation among the groups at any time during the study. Eventhough, dexmedetomidine causes sedation it does not cause any respiratory depression or a change in saturation[28]. In our study, the duration of motor seizure did not vary between the two groups. In group C the mean seizure duration is 36.9 ± 3.5 minutes and in group D 37.8 ± 3.2 minutes. This is important since the duration of seizure coincides with the efficacy of the treatment in ECT. This is consistent with the results of Ravichandra dodawad *et al.*, comparing saline and dexmedetomidine in ECT, (group C-39.56 ±81s, group D - 38.23±90s) and in study of Zakine begec *et al*, both motor and electroencephalography (EEG) seizure duration in the control group (35.65±14.89 and 49.07±9.94 respectively) were similar to that in the dexmedetomidine group (33.30±12.01 and 45.15±17.79 s respectively). In Tarek Shams *et al*, noticed motor seizure duration in ketofol group was significantly less compared to ketofol-dex group (35.8 ± 6.6s versus 38.9 ± 4.9s). In our study, the time to recovery is higher in the dexmedetomidine group (36.8 ± 3.2 min) than the control group (10.6± 2.5 min) with a p value (0.000). Similar results obtained in Fu Wen *et al.*, study the sedation scores on the visual analog acale were increased in the dexmedetomidine group compared to saline group Time to orientation (27± 6 vs 17± 5min) and discharge from the recovery unit after ECT were significantly longer in patients in dexmedtomidine compared to saline group. The agitation score is much lower in the dexmedetomidine than the control group (1.08 ± 0.2 vs 1.40 ± 0.7) with a p value of 0.03 these results consistent with the studies of Tarek Shams et al.,the number of patients with agitation score > 2 was significantly lower in ketofol-dex group (1.4%) compared to ketofol group (8.6%) with a p value of 0.014 and in McGee P.Janey et al , study each patient received 0.75µg/Kg of dexmedetomidine and they had a smooth emergence after the general anaesthesia with a longer recovery period and a stable hemodynamic [8,9]. Our study correlates with the study of Cohen *et al* in which patients with post itcal agitation [10,12] responded quite favourably to dexmedetomidine.

Azaaz U Haq *et al* , studied the effectiveness of Dexmedetomidine for Prevention of Post-Ictal Agitation after Electroconvulsive Therapy, in elderly population(age > 65)

There were no intraoperative complications like bradycardia hypotension, arrythmias during the study in any patients [10,11]. Several studies have highlighted the use of dexmedetomidine 0.5 mcg/ Kg is effective in attenuation of haemodynamic [12,13]. The findings of all these studies were comparable to our study.

One of the limitations of our study is that we did not measure plasma catecholamine levels, which was not feasible in our institute.

CONCLUSION: We conclude that dexmedetomidine at a dose of 0.5µg /Kg given before Modified ECT attenuates the hyper dynamic response associated with the stimulus without any change in the seizure duration. It also decreases the incidence of postictal agitation after ECT with only a slight delay in time for recovery.

REFERENCES

1. Cowen P, Harrison P, Burns T. Shorter Oxford textbook of psychiatry. Oxford: Oxford University Press; 2012.
2. Miller R. Miller's Anesthesia. Philadelphia, Pa: Elsevier, Churchill Livingstone
3. Haq U. Aazaz, Espinoza Randall , Chen Stephen –American J of geriatric psychiatry 22;3-76
4. Coursin DB, Coursin DB, Maccioli GA – Dexmedetomidine.Curr Opin Crit Care 2001;7:221-226.
5. Miller R. Miller's Anesthesia. Philadelphia, Pa: Elsevier, Churchill Livingstone; Pages 991-993.
6. Baddigam K, Russo P, Russo J et al., – Dexmedetomidine in the treatment of withdrawal syndromes in cardiothoracic surgery patients. J Intensive Care Med 2005; 20:118-123.
7. Chrysostomou C, Shiderly D, Berry D et al., – Dexmedetomidine, a novel agent for the acute treatment of supraventricular tachyarrhythmias after pediatric cardiac surgery. Crit Care Med 2007;8:A2
8. Kellner C. Handbook of ECT. Washington, DC: American Psychiatric Press; 1997. 25Rozet I. – Anesthesia for functional neurosurgery: the role of dexmedetomidine. Curr Opin Anaesthesiol 2008; 21:537-543.
9. Frost EA, Booij LH-Anesthesia in the patient for awake craniotomy. Curr Opin Anaesthesiol 2007;20:331-335.
10. Ravichandra Dodawad,G.B. Sumalatha, Sandeep P, ParashuramJajee "Dexmedetomidine at a Dose of 0.6µg/ Kg in Attenuation of Hemodynamic Stress Response of Electroconvulsive Therapy". Journal of Evidence based Medicine and Healthcare; Volume 2, Issue 32, August 10, 2015; Page:4771-4778, DOI: 10.18410/jebmh/2015/669.
11. Mizrak A, Koruk S, Ganidagli S, Bulut M, Oner U. Premedication with dexmedetomidine and midazolam attenuates agitation after electroconvulsive therapy. J Anesth 2009; 23:6-10.
12. Penttilä J, Helminen A, Anttila M et al., – Cardiovascular and parasympathetic effects of dexmedetomidine in healthy subjects. Can J Physiol. Pharmacol 2004; 82:359-362.
13. Paris A, Tonner PH-Dexmedetomidine in anaesthesia.Curr Opin Anaesthesiol 2005;18:412-418.
14. McCarty TM, Arnold DT, Lamont JP et al., – Optimizing outcomes in bariatric surgery: outpatient laparoscopic gastric bypass. Ann Surg 2005;242:494- 498
15. Kobayashi A, Okuda T, Kotani T et al., – Efficacy of dexmedetomidine for controlling delirium in intensive care unit patients. Masui. 2007;56:1155- 1160.
16. Szumita PM, Baroletti SA, Anger KE et al., – Sedation and analgesia in the intensive care unit: evaluating the role of dexmedetomidine. Am J Health Syst Pharm 2007;64:37-44.
17. Jalonen J, Halkola L, Kuttila K et al., – Effects of dexmedetomidine on coronary hemodynamics and myocardial oxygen balance. J CardiothoracVasc Anesth 1995;9:519-524
18. Ma D, Hossain M, Rajakumaraswamy N et al., – Dexmedetomidine produces its neuroprotective effect via the alpha2A-adrenoceptor subtype. Eur J Pharmacol. 2004;502:87-97.
19. Baddigam K, Russo P, Russo J et al., – Dexmedetomidine in the treatment of withdrawal syndromes in cardiothoracic surgery patients. J Intensive Care Med 2005;20:118-123.
20. McGee P.Janey Use of dexmedetomidine for smooth recovery from general anaesthesia after ECT, a case series;Anaesthesiology A844
21. Cohen MB , Stewart JT. Treatment of post electroconvulsive therapy agitation with dexmedetomidine ;Journal of ECT 2013;jun 29(2),e23-24.
22. Begec Z, Toprak HI, Demirbilek S, Erdil F, Onal D, Ersoy MO. Dexmedetomidine blunts acute hyperdynamic responses to electroconvulsive therapy without altering seizure duration. ActaAnaesthesiol Scand. 2008 Feb; 52 (2): 302-6. Epub 2007 Nov. 1.